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BRITISH MEDICAL JOURNAL

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OBSERVATIONS ON THE TREATMENT OF PULMONARY TUBERCULOSIS AT THE PRESENT TIME*

BY

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[WITH SPECIAL PLATE]

In previous Mitchell Lectures the emphasis has usually been on some aspect of tuberculosis as seen through the eyes of a physician engaged in clinical practice. In selecting a subject I was aware that the late Dr. F. G. Chandler talked on the diagnosis and treatment of pulmonary tuberculosis in 1940, but so much has changed in the intervening 14 years with the advent of specific antibacterial therapy and the advances thereby made possible in thoracic surgery that I felt my observations on the present state of therapy in this disease might not be out of place. I shall deal with my own clinical material and with work in this country of which I have at least some slight personal knowledge.

We all admit that preventive medicine does far more in the eradication of tuberculosis than the puny efforts of the clinician in dealing with the sick ; in Scandinavia, certain provinces of Canada, and some parts of the United States it would appear that clinical tuberculosis will disappear in the near future, as smallpox and diphtheria have in Great Britain.

The mortality from respiratory tuberculosis has shown a steady decline for the past century, save for the two world wars, but the fall since 1948 has been striking. From 1931 to 1939 the average total annual death rate for England and Wales was about 25,000 ; from 1940 to 1944 the figure was approximately 22,000, but in 1945 it was 20,000, and by 1953 it was just under 8,000. Whether this abrupt fall is related to antibacterial therapy is for the statistician to decide. The notification of new cases remains high—in the neighbourhood of 50,000 per annum since 1947—but has dropped from about 48,000 in 1952 to 41,000 in 1953. The number of non-notified deaths from respiratory tuberculosis is still high, even though it has fallen from 3,530 in 1937 to 2,239 in 1952 and 1,751 in 1953. Papworth has shown us that in the known case simple hygiene to prevent spread of infection is highly efficacious ; it is the undiagnosed positive case which is the danger.

With mass radiography and improvement in the standard of chest clinics, the type of case seen at a London chest hospital has altered appreciably of late years, but the management of the symptomless case remains a real problem. Apart from cases found by mass radiography, pulmonary tuberculosis seems to be changing its character, and we know that over 45% of probationer nurses and first-year medical students are Mantoux-negative when they first attend hospital.

*Based on the Mitchell Lecture delivered before the Royal College of Physicians, London, June 18, 1954.

Bed Rest

In recent years one of the greatest advances has been the Medical Research Council's trials of chemotherapy, with simultaneous clinical and scientific observations on large numbers of patients with similar types of disease, using control cases, and with a panel examining results dispassionately. For this we owe much to the late Dr. Marc Daniels. In the first streptomycin trials in 1947–8 the control cases were given six months' proper bed rest, and the improvement in a proportion of these was impressive. Dr. Wallace Fox, in a personal communication, has told me that 17 of the original 52 control (acute bilateral) patients are still alive after five years ; most of these were not given chemotherapy for the first two years, although many had collapse measures.

In my experience, in the newly diagnosed case the incidence of "extension" of disease during the period of a few weeks while the patient is awaiting admission to hospital is alarming. Explicit and simple instructions on the details of bed rest at home must be given to the patient. My directions are that he must have the foot of the bed raised on two bricks and use no more than two pillows ; he is allowed up once a day only, for toilet purposes and to wash and shave, otherwise he must lie in bed throughout the twenty-four hours, including meal-times, save for using a chamber for emptying the bladder.

In cavitated disease, postural retention as practised extensively by Dillwyn Thomas at Sully Hospital, Glamorgan (Thompson, Savage, and Rosser, 1954), with the patient lying in a simple plaster cast and with the foot of the bed raised, at the correct inclination for six to twelve weeks, is a real advance in treatment, especially when combined with chemotherapy. Although cavities rarely close permanently, they usually decrease markedly in size and the surrounding exudative disease becomes more proliferative. Cough and sputum disappear quickly as a rule. The lung is then in a better state for further treatment and less extensive surgical measures are required.

Antibacterial Therapy

The discovery of streptomycin by Waksman and his colleagues (Schatz, Bugie, and Waksman, 1944) and the experimental work and clinical trials of Hinshaw and his coworkers (1944, 1945, 1946) have revolutionized the treatment

of pulmonary tuberculosis, not only as regards medical treatment, but by allowing more extensive surgical measures by collapse or resection with a greatly diminished risk of broncho-pleural fistula and dissemination of the disease. Another advance was the discovery that the combination of *p*-aminosalicylic acid (P.A.S.) with streptomycin materially decreased the incidence of resistant strains of bacteria to both drugs. In 1951 isonicotinic acid hydrazide (isoniazid) was discovered. This powerful, cheap, and relatively simple drug can be taken by mouth, and again the incidence of resistant organisms is diminished when it is combined with either of the other drugs.

Obviously new and possibly more efficient drugs will be produced; the clinician tends to become confused with the rapid changes taking place these days. The principle of testing under scientific control the value and dangers of new therapeutic agents is of the greatest value, and it behoves us not to discard a well-tried combination of drugs until there is good evidence that a new combination is more efficient and less toxic.

On looking back over the milestones in therapy during the past 30 years—insulin, liver extract, sulphapyridine, penicillin, and cortisone in Addisonian crisis—I still treasure the moment of doubting amazement when I saw the results of streptomycin treatment in the group of young patients with acute exudative disease who were in the Brompton Hospital during the Medical Research Council's trials from 1947 to 1953. The following is an example of such a case.

Case A.—A girl aged 18 years was admitted to the Brompton Hospital in January, 1949, with four months' history of cough, night sweats, and loss of weight. She was acutely ill with tuberculous bronchopneumonia: temperature, 103° F. (39.4° C.); pulse, 120; respirations, 30. She was given streptomycin, 1 g. daily, with P.A.S., 15 g. daily for 14 weeks. In January, 1950, a left pleuropneumonectomy was performed, and she was subsequently sent to a sanatorium for six months. She is well and working four years later. (Plate, Figs. 1–3.)

When participating in the treatment of this girl I was thrilled by the vista of what might be accomplished in the future treatment of pulmonary tuberculosis. Such a prospect, coupled with the advent of lobar and segmental resection after chemotherapy, may have blinded us to the value of the older and tried forms of treatment, however. There is a tendency in some countries to accept the view that the proper treatment of pulmonary tuberculosis is a prolonged course of rest with chemotherapy followed by the resection of the remaining areas of nodular caseous disease. The rather scathing term "pimplectomy" has been used by some clinicians who feel that the pendulum of resection has swung too far.

Thoracic surgery has had an immense influence in giving us knowledge of pathological processes in the lungs at an earlier stage than is seen at necropsy. As a class, thoracic surgeons are highly trained and very able; they handle the lungs in life and can correlate the *x*-ray appearances with the actual lesions found. It is therefore not surprising that the thoracic surgeon should have great influence in advising in and organizing the treatment of pulmonary tuberculosis. Perhaps it is not untimely to suggest that we physicians, in publishing the results of medical treatment, should use the same factual presentation as our surgical colleagues.

In the treatment of pulmonary tuberculosis we have three main weapons in addition to the long-established principles of rest, diet, fresh air, and graded exercise: (*a*) antibacterial drugs without collapse therapy; (*b*) collapse treatment, either medical or surgical, in which the diseased lung remains in the body and possibly acts as a stimulus to antibody formation; and (*c*) resection of the diseased part, which may act as a reservoir of subsequent infection. Any of these three may be used singly or in combination. We must be clear as to our aims in treatment, which are: (1) permanent healing of the lesions; (2) minimal loss of respiratory function; and (3) the attainment of these in the shortest time. The treatment of tuberculosis is long in any case, and *unnecessary* months of idleness may have serious effects on morale and on domestic happiness.

Artificial Pneumothorax Therapy

I want to review our results by medical collapse measures alone, before antibacterial therapy was introduced, so as to have a base-line for the comparison of treatment to-day. The true value of artificial pneumothorax therapy is difficult to assess from the voluminous literature, because of variations in the classification of the extent of disease and an often inadequate follow-up. A paper by Foster-Carter, Myers, Goddard, Young, and Benjamin (1952) on cases in the Frimley Sanatorium furnishes some sort of yardstick. The paper covers the five-year period 1937–42 and 1,153 patients, of whom 955 had cavitated disease, and approximately 99% were followed up for at least eight years. Classification was according to the number of zones in the postero-anterior radiograph on admission; this was shown by Hartley, Wingfield, and Burrows (1935), on their experience of 2,759 cases at Frimley, to be probably the most reliable classification on which to base prognosis. It is as follows: Class I, not more than one zone involved; Class II, unilateral or bilateral disease not involving more than three zones; and Class III, four or more zones involved.

The average period of stay in the Brompton Hospital was 40 days and in Frimley Sanatorium 140 days, amounting to about seven months' institutional treatment in all. No doubt the criticism could be made that these cases are a selected group, as Frimley did not take bed-rest cases, and that Class III (advanced) cases diagnosed radiologically were not really so "advanced" as those transferred to other sanatoria where bed-rest patients were accepted. Obviously a ten-year follow-up would have been better than one of eight years. Be that as it may, the figures show a fair proportion of Class III cases. A further point is that these patients were under the control of different physicians at the Brompton Hospital, whose opinions varied widely as to the use of collapse measures, division or not of adhesions, and duration of treatment. This paper is, however, impressive in that the numbers are sufficient, with nearly 99% of cases followed up for eight years, and the results statistically significant. I am grateful to the authors for allowing me to quote from it.

Of 256 patients with cavitated disease, who had *no collapse therapy* at any time, 113 (44%) survived eight years; 93 (82%) of these were working and 62 were known to have a negative sputum. Of 83 patients in Classes I and II, 74% survived eight years, and of 173 in Class III, 33% were alive after that period. Thus it is clear that the extent of the original disease is of more importance than the size of the cavities. Of the 616 cases of cavitated disease reported in the much-quoted paper by Barnes and Barnes (1928), 130 were followed up for five years; of these, 79% were in Class III and 85% had large cavities; the five-year survival rate was only 10%. The Frimley figures give a less gloomy picture: of 104 Class III patients with large cavities 33% survived five years and 23% eight years.

In non-cavitated disease artificial pneumothorax treatment appears to be of little benefit. Of 130 patients treated without collapse therapy 88.9% survived eight years, although 24 (18%) required collapse therapy later. Of 62 patients treated by unilateral pneumothorax, 87% survived eight years and 35% needed further collapse. It is possible that the physician selected the less favourable cases for collapse therapy. The point I want to emphasize is the relatively good prognosis in non-cavitated disease without antibacterial therapy.

Of 457 patients with cavitated disease treated by unilateral artificial pneumothorax 316 (69%) survived eight years; 423 (93%) of these cases came into Classes II and III. An eight-year survival rate of 82% was obtained in 240 cases in Class II, and in 48% of 183 cases in Class III.

The striking finding of the Frimley investigation—in my view a most unexpected one—is the good result attained by

what I would call "contra-selective collapse." The authors define three grades of collapse resulting from artificial pneumothorax: (*a*) "satisfactory"; (*b*) "adherent satisfactory," with disappearance of the cavity; and (*c*) "adherent unsatisfactory," in which the cavity fails to close within one year. A further surprising fact is that extension of disease to the opposite lung occurred in 25% of the "satisfactory" group, 23% of the "adherent unsatisfactory" group, and only 11% of the "adherent satisfactory" group. This might be just a chance happening and is difficult to explain on other grounds.

Empyema occurred in 17% of the whole series, with 14% in the "satisfactory" group, 9% in the "adherent satisfactory" group, and 36% in the "adherent unsatisfactory" group, which again shows the danger of maintaining treatment when the cavity fails to close. The results eight years after termination of the pneumothorax show that 64% of the "satisfactory," 60% of the "adherent satisfactory," and about 28% of the "adherent unsatisfactory" group were well and working.

The deductions I draw from this important paper are as follows: (1) The grading according to zones in the radiograph gives a good indication of prognosis. (2) A satisfactory collapse in which the cavity closes gives much better results than are obtained without collapse therapy. (3) An artificial pneumothorax which fails to close a cavity in a reasonable time is dangerous. (4) The incidence of empyema is 17%, which is too high; it occurs particularly in the group with persistent cavities.

I have analysed a series of my own cases, 62 in all. So far as I am aware this includes every case admitted to the Brompton Hospital under my care in which I recommended pneumothorax treatment during the six years 1942–7 inclusive; these cases are therefore not included in the Frimley report. The series is too small to be of any statistical value, and in addition seven patients have not been traced.

The results, which are analysed in Table I, were good in 36 and bad in 11, and there were 10 deaths. I believe in attaining an anatomically perfect collapse where possible, and adhesion section was performed in 35 out of 47 cases,

TABLE I.—*Results of Artificial Pneumothorax in Cases Admitted to the Brompton Hospital During* 1942–7

	Non-cavitated, Sputum Negative	Class I	Class II	Class III	Total
Failed A.P. ..	—	—	5 (1 died)	2 (1 died)	7 (2 died)
Lost touch ..	—	1	5	1	7
A.P. maintained ..	—	1	—	—	1
Result good ..	7	11	11	7	36
Treatment failed ..	—	—	6 (5 died)	5 (3 died)	11 (8 died)
Total ..	7	13	27	15	62 (10 died)

Years after termination of A.P.	Good Results				
Over 4	7	6	6	5	24
3 to 4	—	2	4	1	7
1½ to 3	—	3	1	1	5
Total ..	7	11	11	7	36

yet empyema occurred in only 2 of the 47 patients, who were all followed up for at least eighteen months after termination of the pneumothorax. In both cases a suspended cavity was obvious and the pneumothorax should have been terminated within a few weeks. I can also endorse the observation by F. H. Young (1949) that cases under the care of one physician throughout do better than those under multiple control.

Phrenic Paralysis with Pneumoperitoneum Treatment

Banyai (1946) introduced this treatment about 1934 and is enthusiastic as to its value. Many papers on its application have been published, but in this country at any rate it is employed chiefly as a preliminary treatment for acute exudative disease, to give some rest to the lung before more definite collapse measures are undertaken. I have used it in a limited number of (generally advanced) cases since 1942, and feel that its value, particularly when it is combined with chemotherapy, is perhaps not sufficiently appreciated. I have had no experience with pneumoperitoneum without phrenic paralysis.

As in pneumoperitoneum the pleural surfaces are not separated, the complications seen with artificial pneumothorax—such as fluid, empyema, haemothorax, and frozen lung—do not occur. Serious complications are rare—less than 5%. As with pneumothorax treatment, a certain amount of skilled supervision is required to attain the objective aimed at—relaxation and rest of the lung selected for phrenic paralysis. This entails a careful search for an accessory phrenic nerve at the time of the phrenic crush and suitable refills to keep the paralysed diaphragm high in the chest, to the level of the third rib or intercostal space in front as seen in the ordinary postero-anterior radiograph; also recrushing the nerve if the hemidiaphragm recovers too early. Careful assessment is needed at a suitable time, usually between six and nine months, to ascertain whether the aims of treatment, especially cavity closure, have been attained; if not, further treatment should be instituted. Lateral radiographs and also tomographs are necessary to decide the progress of the lesion, as pneumoperitoneum affects mainly the posterior part of the lung.

All but two of my 68 patients who have undergone this procedure at the Brompton Hospital have been followed up to date. Apart from four cases in which the pneumoperitoneum was induced by other physicians but supervised by me, the treatment has been given on my recommendation in all cases. Treatment has been abandoned for at least eighteen months, and no patients have been given chemotherapy as part of their treatment. My registrars, Dr. K. J. Gurling and Dr. W. H. Helm, have reviewed the case sheets and original radiographs with me, so as to obtain an unbiased assessment of the extent of the original disease and the results of treatment. When the original films were not available, owing to the fire at the Brompton Hospital in 1950, we have taken the radiologist's report or my own notes on the original films.

Table II gives the over-all results obtained in the 68 cases, divided into Classes I, II, and III and "lower-lobe cavities." In 32 the results were classified as "good," in 20 as "failed,"

TABLE II.—*Over-all Results in* 68 *Cases Treated by Phrenic Paralysis and Pneumoperitoneum*

Class	No. of Patients	Results of Treatment				Received Other Treatment	Final State			
		Good	Failed	Non-proven	Lost		Well and Working	Disease Progressive	Died	Lost
I ..	6	3	1	2	—	3	6	—	—	—
II ..	24	10	9	4	1	7	18	1	4	1
III ..	26	10	7	8	1	12	16	2	7	1
Lower-lobe cavities	12	9	3	—	—	3	9	2	1 (non-tb)	—
Total	68	32	20	14	2	25	49	5	12	2

and in 14 as "non-proven"; 2 have been untraced for one and a half years. With further treatment in 25, 49 of the patients are well and working and have negative sputum. Table III gives a detailed analysis of 65 cases.

The 6 cases in Class I need little comment. In Case 1 the patient, a business man, had to travel by air, and a thoracoplasty was therefore performed. In Case 4 an artificial pneumothorax was induced because the physician did not believe in pneumoperitoneum treatment. In Case 6 a

TABLE III.—*Detailed Analysis of the 65 Cases Treated by Phrenic Paralysis with Pneumoperitoneum*

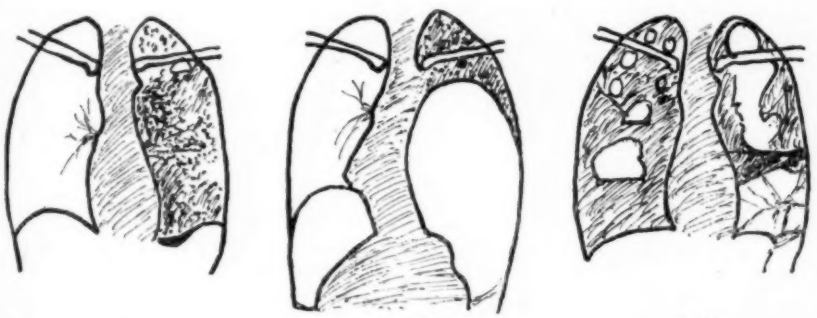
Case No.	Age and Sex	Standard of Treatment	Duration (Years)	Result	Years After Termination	Further Treatment and Remarks	Final State
				CLASS I			
1	31 M	Good	5/12	Non-proven	3½	R. thoracoplasty	Well
2	29 M	Poor	3½	Good	1		"
3	25 M	Good	5	"	1½		"
4	26 M	Poor	9/12	Non-proven	7½	R. artificial pneumothorax with fluid	"
5	20 F	Good	3½	Good	1		"
6	44 F	Fair	4/12	Failed	2½	L. plombage	"
				CLASS II			
1	40 M	Good	1	Non-proven	9½	Pneumoperitoneum added to R. artificial pneumothorax. Subsequently relapsed more than once. Working 1953	Well
2	18 M	Poor	4/12	Good	11	Well 10 years. ? Smouldering disease 1 year	
3	18 M	Fair	4½	"	5		"
4	29 M	Good	2½	"	6		"
5	20 M	Poor	8/12	Non-proven	8	Extensive bilateral infiltration with R. pleural effusion	"
6	16 M	"	3½	"	½	Left sanatorium when radiograph satisfactory. Did not attend chest clinic. Died suddenly	Died
7	34 M	Very poor	2½	Failed	2½	Died. Cavities closed temporarily. Should have had thoracoplasty	
8	27 F	Good	3½	Good	3½	L. artificial pneumothorax abandoned before pneumoperitoneum started	Well
9	39 M	Poor	2	"	4		"
10	45 M	Good	10/12	"	6½		"
11	27 F	"	4½	"	2½		"
12	22 F	"	4½	"	2		"
13	27 F	Fair	1½	Failed	4½	Pneumoperitoneum abandoned for ulcerative colitis after 18 months. R. thoracoplasty	"
14	18 F	Good	4/12	"	5½	R. thoracoplasty	"
15	20 F	"	3/12	"	4½	Cavity bigger with pneumoperitoneum, but closed with artificial pneumothorax. Abandoned 1½ years	"
16	38 F	Poor	3	Good	2½		"
17	37 M	Good	5/12	Non-proven	4½	Sputum positive at 5 months. Bilateral artificial pneumothorax maintained	"
18	34 F	"	1½	Failed	1	Died. Cavity closed 1 year but reopened. R. thoracoplasty. Died 1 year	Died
19	21 M	Fair	1½	"	3½	Difficult patient: ceased treatment. Bilateral disease. Long chemotherapy	Well
20	41 M	Good	1½	"	2½	L. thoracoplasty. Prolonged chemotherapy. Sputum positive 2 years after	"
21	30 M	"	7/12	?	2	Abandoned on ship after 7 months	Lost 2 years
				CLASS III, GROUP A (with Aim of Cure without other Treatment)			
1	18 F	Good	4½	Good	5		Well
2	21 M	"	2	Failed	6	R. thoracoplasty 1948. L. artificial pneumothorax 1950–3	"
3	24 M	"	½	Non-proven	7½	Bilat. artificial pneumothorax 4 years. R. upper lobectomy 1952	"
4	44 M	Fair	1½	Failed	4½	R. thoracoplasty 1949	"
5	19 F	Poor	3½	Good	1½	Chemotherapy, 42 g. Streptomycin	"
6	20 F	Good	½	Non-proven	3½	L. artificial pneumothorax. Persists 1954	"
7	19 F	"	4½	Good	3	L. thoracoplasty 1951. Sputum positive	"
8	26 M	Poor	6	Failed	6	R. thoracoplasty 1953. L. artificial pneumothorax maintained	Fair
				CLASS III, GROUP B (with Aim of Curing One Side)			
1	46 M	Good	2	Failed	2	Died 1947. Alcoholic	Died
2	21 F	Poor	4	Good	4½	R. plombage 1949	Well
3	53 M	"	4/12	Failed	3	Died 1949. Disliked refills. Downhill course after pneumoperitoneum stopped	Died
4	28 F	Good	1½	Good	6	R. thoracoplasty 1948	Well
5	35 F	Fair	5/12	Failed	½	Died 1948.* Gross cavitated disease. Enteritis 6 months	Died
6	22 F	Poor	4½	Good	3½	R. thoracoplasty 1951. Sputum negative	Well
7	27 M	Good	1½	Failed	1½	Died 1951. L. artificial pneumothorax plus empyema	Died
8	20 F	"	3	"	1½	Deteriorating	Ill
				CLASS III, GROUP C (preparatory to Surgical Treatment of Same Side)			
1	22 F	Good	8½	Good	1½	R. artificial pneumothorax. Empyema. Wash-outs	Well
2	30 F	Fair	1½	Failed	½	L. thoracoplasty 1946. Died 1947	Died
3	28 F	Good	1	Good	6	Abandoned treatment after one year	Well
4	63 M	Fair	½	Non-proven	7½	Slowly progressive	Ill
5	37 M	Very poor	1½	"	—	Died. Treatment poor	Died
6	34 M	" "	1½	"		Treatment very poor	Lost 1½ years ?Dead
7	20 F	Fair	½	Failed	6½	Cavity bigger. L. thoracoplasty 1949	Well
8	23 M	Good	1½	Good	5½	R. thoracoplasty 1948. Spread L. in 1953	"
9	31 M	Poor	6/52	Non-proven	3	Died 1951. Disease progressive	Died
10	22 M	Good	2½	Good	3	R. artificial pneumothorax. L. thoracoplasty 1951	Well
				LOWER-LOBE CAVITIES			
1	45 F	Good	½	Good	5	Patient stopped refills at 6 months. Well April, 1954	Well
2	20 F	"	4	Failed	5	Cavity blocked: reopened at 4 years. Lobectomy Feb., 1949	"
3	27 F	"	4½	Good	4	Also large cavity R. apex. Phrenic evulsion	"
4	15 F	Poor	5½	"	2½	L. artificial pneumothorax 5½ years	"
5	27 M	"	8/12	Failed	7½	Pneumoperitoneum abandoned too soon. L. extrapleural artificial pneumothorax. Mixed empyema. R. thoracoplasty 1949. Sputum positive	"
6	20 F	Good	1½	Good	7	L. artificial pneumothorax failed; pneumoperitoneum added and cavity closed. R. artificial pneumothorax for cavity. Fresh disease L. upper lobe after 1½ years	"
7	30 F	"	1½	"	3½	Haemoptysis persisted. Sputum negative. R. lower lobectomy Nov., 1948. Cavity closed. Died acute enteritis 1952	Died
8	38 M	Fair	3	Failed	4½	Patient abandoned refills. Cavity reopened May, 1950, with spread to R. lower lobectomy Jan., 1951—further disease L. upper lobe. Chemotherapy	Fair
9	16 F	Good	5	Good	2½	Round foci on R cavitated. L. lower lobectomy Nov., 1951. Cavity well shut off; R. still smouldering	"
10	35 F	"	5	"	2½	Lower-lobe cavity healed. Smouldering L. apex—plombage 1953	Well
11	16 M	Fair	2½	"	5½		"
12	16 F	Good	3½	"	1		"

pleural effusion occurred before treatment, with the formation of a thick-walled apical cavity. In Class I the results were : 3 good, 2 non-proven, and 1 failed.

The 21 cases in Class II were treated with the aim of cure without further treatment. The results were : good, 9; failed, 7; non-proven, 4; and untraced, 1. The final state is more cheerful in that after treatment 17 patients are well and working, 3 have died, and the other is untraced. The remaining 3 cases of the 24 in Class II were treated in preparation for surgical treatment on the same side, and are therefore not included in Table III. The first patient had a left plombage in 1951 and is now well; the second, a diabetic girl of 27 years with extensive bilateral cavitation, has had a left extrapleural pneumothorax and a right plombage, but is going downhill. The history of the third case is as follows :

Case B.—The patient, a woman of 39 with chronic unilateral cavitated disease, developed a tuberculous bronchopneumonia following a large haemoptysis and the left lung was virtually destroyed. Streptomycin treatment had to be stopped after a fortnight owing to the development of severe toxic reactions to the drug. With a left phrenic crush and pneumoperitoneum the diaphragm was kept high and the disease controlled. Owing to the presence of disease on the right side it was necessary to wait for eighteen months before a left pneumonectomy could be performed, but during most of this time she was free of cough and sputum and doing her housework, and by the end of the period she had regained her normal weight. Unfortunately, when on holiday she was given a refill at a strange clinic, where she had a syncopal attack. This was thought to be due to an air embolism, and the refills were abandoned forthwith without any consultation. The patient died three months later with enormous cavities on both sides. Diagrams of the radiographs are shown below.

The above case illustrates the dangers of dual control, and, moreover, shows the importance, when surgical treatment has been decided upon, not to abandon the pneumoperitoneum until the patient is actually in a surgical bed.



Diagrams of radiographs in Case B. (*a*) Condition before treatment (October 20, 1950): grossly diseased cavitated left lung. (*b*) During treatment (May 4, 1951): left diaphragm up to level of second rib; patient without symptoms, doing full day's housework. (*c*) Condition on day of death (October 21, 1952), three months after treatment had been abandoned: "destroyed" left lung; gross cavitated disease in right.

In Class III, the advanced cases, there are 26 patients who offer a challenge to the clinician for any form of therapy. The aim in these cases was to see how many can be salvaged rather than successfully treated. With chemotherapy, of course, the outlook is better, but these patients did not have this help. I divided them into four groups, as a bald statement of success or failure does not give a fair picture of what may be done : Group A—with the aim of cure without other treatment; Group B—with the aim of curing one side preparatory to collapse or resection on the opposite side; Group C—with the aim of temporary treatment preparatory to collapse or resection on the same side; and Group D—the pneumoperitoneum is maintained indefinitely, as the condition of the patient or the extent of the disease does not allow of major surgery.

In Group A there are 8 cases : 3 were successful, 3 failed, and 2 were non-proven. After further treatment all patients are well and working. In Group B 3 of the 8 cases were successful, the treated lung standing up to surgical collapse of the contralateral side; the other 5 were failures. In Group C, of 10 cases, 4 were classed as successful; 2 of these patients recovered without further treatment. There were 4 non-proven cases and 2 failed. Three cases required thoracoplasty. As in all of the 68 cases the pneumoperitoneum has been abandoned for eighteen months or longer there are no cases in Group D.

An example of the few cases in which a permanent pneumoperitoneum has been maintained is described below. (See Plate, Figs. 4–6.)

Case C.—A man aged 28 years with severe diabetes, Kimmelstiel-Wilson syndrome, and gross hypertension. The extent of the disease and subsequent progress are shown in the radiographs. He has never had antibacterial therapy. For some four years he has been clinically well, without cough or sputum, and is earning his living. I have refused the repeated requests of his chest physician to subject this patient to a thoracoplasty to close the cavity on the left side.

The last part of Table III shows the results in 12 patients with lower-lobe cavities, which are notoriously difficult to close; 3 of these had had unsuccessful pneumothorax therapy previously. Results were successful in 9. In Case 7 the patient died of acute enteritis four years after a lobectomy, performed because haemoptysis persisted in spite of pneumoperitoneum treatment. The resected specimen showed that the original cavity had closed and the bleeding had been caused by an area of bronchiectasis.

The patient in Case 2, a girl of 20, had a large cavity which became "blocked"; she remained well and free of sputum for four years with the pneumoperitoneum, but then "spread" occurred near the cavity and a left lower lobectomy was performed. Five years later she is well and working, having had two children in this time. This case taught me that when a cavity becomes "blocked" but does not progressively decrease in size, resection should be carried out, say after a year.

In Case 5, a male aged 27, the pneumoperitoneum had been abandoned too soon, as clinical and radiological examination indicated that healing was taking place. Subsequently the patient went through a whole series of surgical operations.

Plombage

The late Dr. F. G. Chandler (1940) has said that the thoracoplasty is "a terrific and mutilating operation, one that offends the aesthetic sense and craftsmanship of the surgeon." With the modern thoracoplasty few would now subscribe to this view, particularly as most surgeons use general rather than local anaesthesia, which was not the rule some years ago. The mortality is now less than 5% with experienced surgeons, but the immediate post-operative pain and the common complications, such as atelectasis of the lower lobe and fluid formation, call for fortitude from the most courageous patient. The waiting period for the second stage, and occasionally the third, calls for even more courage. In my experience thoracoplasty still gives most reliable and lasting results, and patients may be seen attending for an annual check, year after year, in good health, in full work, and with minimal deformity. In spite of these good results, however, one feels at times that one is using a sledgehammer to crack a nut.

Like Chandler I had hoped that some form of plombage with a non-irritant substance would be devised, and that—although it contravenes a principle of surgery by leaving a foreign body in the tissues—something better than the old wax plomb would be found.

Wilson in 1948 showed in animals that "lucite" spheres caused little irritation or reaction in the tissues. Trent, Moody, and Hiatt in 1949 published a damning report on

51 cases in which lucite spheres were introduced into the extrapleural space. Woods, Walker, and Schmidt (1950) in the United States, and Lucas and Cleland (1950) in this country, have reported favourably on the use of extrafascial stripping as for a Semb thoracoplasty and then introducing lucite balls (at first hollow, but now solid and sterilizable by boiling), of which they have had considerable experience.

Mr. W. P. Cleland, who has carried out the plombage in most of my cases, has kindly let me have his results to date in 186 of his own cases treated three or more years ago, starting about 1948. Of these only about 56% were good risks and some 12% were designated as "scrapheap" cases. His figures are as follows :

	No. of Cases	
Good results without further treatment	104	134
" " with " "	30	(72%)
Unsatisfactory:		
Persistent cavitation	8	
Active disease	3	15
Sinuses, etc.	4	
Untraced	18	
Died (from unrelated causes, 8)	19	
Total	186	

There were 13 cases with space infection. Of these, 2 were pyogenic ; one had a thoracoplasty and the other was treated by general and local chemotherapy ; both patients are now well. The other 11 cases had late tuberculous infection, the majority with sterile pus ; of these, 2 died from conditions unrelated to their infection, 3 had sinuses, and 6 are well.

Table IV gives details of the 27 cases for which I recommended lucite plombage. The results were good in 20 and moderate in 2, and there were 5 failures. Of the 3 deaths, one patient (Case 7) died from an anaesthetic preliminary to a pneumonectomy, one (Case 22) from epilepsy when cured of her tuberculosis, and the other (Case 23) from carcinoma of the bronchus. Complications were not serious.

It is still early, the longest period since operation being five years, to be sure that the foreign body in the chest will not give trouble, but I am hopeful. From the patient's point of view the advantages of this operation are threefold : (1) it is a one-stage procedure ; (2) it is not followed by chest deformity ; and (3) it has a relatively low complication rate with absence of post-operative shock and severe pain in the majority. From the point of view of the physician there is a fourth advantage—the patient can have the operation in a "medical" ward and so avoid the wait of six to twelve months for a surgical bed.

Resection

In some countries, particularly the United States, resection has become the accepted form of treatment after a long course of antibacterial therapy, varying from six months to two or even three years. Its use may be considered broadly under two headings : (*a*) *direct* therapy, in which the diseased and usually functionless portion of lung, which has previously caused symptoms, is removed, thus eliminating a reservoir of future infection ; and (*b*) *prophylactic* therapy, by which I mean removal of parts of damaged lung not at the time causing symptoms but which are considered to be potential sources of infection at a later date. Certain discrete round foci or bronchiectatic segments of the upper lobes are examples. In regard to direct therapy, the older school argue, from previous experience of collapse therapy, that the diseased lung left in the chest acts as a stimulus to specific immunity, while the younger school have greater regard for the theory of removing "the factory." Controversy over prophylactic resection of apparently benign lesions still continues.

From my own impressions as opposed to factual data I regard bronchiectasis in the upper lobe, collapsed by disease or therapeutic procedures, as a benign condition in all but a small minority of cases, and bronchography shows that it is very common. According to Clegg (1953) discrete round foci may be of three types: (*a*) small areas of tuberculous pneumonia ; (*b*) cold abscesses within the bronchi ; and (*c*) blocked cavities.

In this country Ronald Edwards (Bickford *et al.*, 1952), of Liverpool, who published his first 200 results in 1950, and later Dillwyn Thomas (Thompson *et al.*, 1954) of Sully

TABLE IV.—*Results of Extrafascial Apicolysis with Lucite Plombage*

Case No.	Age and Sex	Complications	Sputum After	Result	Time After Operation (Years)	Further Treatment	Final State
			CLASS I				
1	19 M	Cavity patent. Closed 3/12 posture	Nil	Good	3½	3/12 postural treatment	Well
2	57 M	Small effusion	Negative	"	4	—	"
3	37 F	—	"	"	3½	—	"
4	38 M	—	"	"	2½	—	"
5	44 F	—	Positive (culture)	"	2½	—	"
6	21 M	—	?	"	1½	—	"
			CLASS II				
7	28 M	—	Positive	Failure	1½	Anaesthetic death at operation for R. pneumonectomy	Dead
8	19 F	—	Negative	Good	4½	—	Well
9	32 F	Fluid: one aspiration	"	"	5	—	"
10	26 F	—	"	"	4½	—	"
11	22 M	Pyogenic empyema	"	Failure	4½	R. thoracoplasty	"
12	37 M	Fluid and atelectasis. Thrombosis arm vein	Positive	"	3½	L. upper lobectomy	Well. Sputum positive
13	40 F	Fluid: four aspirations	Nil	Good	4½	—	Well
14	29 M	Atelectasis few days	?	"	1½	—	"
15	41 F	Nil / Nil bilateral	Negative	"	L. 1½ / R. 1	—	"
16	21 M	Pneumothorax. Haemorrhage into space	"	"	1½	—	"
17	44 F	Fluid: two aspirations	"	"	1½	—	"
18	30 M	" " — "	"	"	1½	—	"
19	30 F		"	"	1½	—	"
			CLASS III				
20	27 M	Pneumothorax and atelectasis. Fluid: two aspirations	Negative	Good	5	R. artificial pneumothorax 5 years. Abandoned 1 year	Well
21	21 F	Fluid: two aspirations	Positive	"	5	Plombage repeated June, 1950	"
22	25 F	—	"	Fair	2½	R. pneumonectomy Dec., 1950	Died of epilepsy
23	40 M	Fluid: two aspirations	"	Failure	1½	—	" " carcinoma
24	33 F	—	"	Fair	3	—	Well
25	35 F	Haemothorax: one aspiration	Negative	Good	2½	—	"
26	68 M	—	Positive	Failure	1½	—	Fair. Sputum positive
27	28 M	Pneumothorax: atelectasis R.	Nil	Good	1½	—	Well

Hospital in Wales, with their assistants have performed a large series of resections, although most thoracic surgeons are undertaking a greater or less number according to their views on the pathogenesis and progress of tuberculosis of the lungs. The tendency now is that with lobectomy or resection of more than one segment of a lobe it is probably wise to prevent over-inflation and possible "reactivation" in the remaining lung by some operation to reduce the size of the hemithorax. This may be done at the time of the original resection or at a later date; in either case it adds something to a simple thoracotomy.

Mr. Edwards has kindly allowed me to quote these figures of his results. Of 1,023 patients, 20 died within three months of operation, an immediate mortality of 1.95%. In a four-year follow-up of 112 cases there are 61 pneumonectomies with 4 deaths and 4 with late exacerbations of disease; 49 lobectomies with one post-operative death and 5 exacerbations; and 2 segmental resections with 1 exacerbation. After four years 95% of the 112 patients are well and working.

By 1952 the percentage of segmental resections performed had increased to approximately 66% of all resections. Up to March, 1952, after 259 segmental resections, only one post-operative death and two empyemata had occurred.

Thompson and Savage, working with Dillwyn Thomas at Sully Hospital, report for the five-year period to June, 1953, 266 cases of resection of various types with an operative mortality of 1.88% and "apparently arrested" disease in about 97%. It is too early yet to assess final results.

For what it is worth I present my own small series of resections. By nature I am conservative, and while welcoming advances in treatment I am inclined to wait and see the results of innovations in the cases of more enterprising physicians. Of 11 pneumonectomies, mostly with "destroyed" lung, results were good in 9. There were 2 deaths, one from acute bronchitis 2½ years after, the other from epilepsy one year after; the latter has already been referred to under "Plombage." Of 21 lobectomies, results were good in 18 and poor in 2; there was one failure. Only 4 had post-operative collapse to decrease the size of the hemithorax. Of 8 segmental resections, results were good in 6 and bad in 2.

Conclusion

In this paper I have given certain figures, which I believe to be reliable, concerning artificial pneumothorax and pneumoperitoneum treatment without antibacterial therapy. Now that antibacterial therapy is available the results of surgical intervention are much better, as also are those of medical collapse. With medical collapse and chemotherapy, in my experience, many patients with minimal or moderate disease can safely return to their homes and occupations within eight to twelve months.

In the Frimley series from 1937 to 1942 quoted above the incidence of empyema was 17%—an appalling figure. If this is the true risk at the present time, then it is no wonder that artificial pneumothorax is much less used than in the past. The results in suitable cases five years after refills have been discontinued are very good, however. Of my own series of 62 patients treated with artificial pneumothorax from 1942 to 1947 it is possible, but I think unlikely, that in each of the seven untraced cases an empyema developed. Of 47 cases followed up for at least eighteen months after the pneumothorax was abandoned, only in two did empyema develop; both of these were badly selected cases in which treatment should have been abandoned forthwith.

I now teach my students that the indications for artificial pneumothorax are very limited and that the treatment should be used only in the following circumstances: (1) when the clinical duration of disease is at least four months and chemotherapy has been given for a further four weeks; (2) when cavities are less than 4 cm. in diameter; (3) when there is no clinical or radiological evidence of endobronchial disease; and (4) when it is estimated that after the lung has healed it will fill the hemithorax without risk of over-inflation or displacement of the mediastinum. I further impress upon them that they should not hesitate to abandon an artificial pneumothorax that has been started if conditions are unfavourable.

We physicians unfortunately often deal with advanced cases quite unsuitable for pneumothorax or surgical treatment at that stage. In these cases the use of chemotherapy with postural retention for two to three months, followed by phrenic crush and pneumoperitoneum, will give an idea of future possibilities. A plombage on the worse side at the appropriate time may in some of these rather hopeless cases enable the patient to get back to a useful life within twelve to eighteen months. It is perhaps not sufficiently appreciated at the present time that pneumoperitoneum with phrenic paralysis added to antibacterial therapy has real value.

The following is a typical case (see Plate, Figs. 7–9).

Case D.—An officer in the Merchant Navy aged 27 years had been a contact of his brother and sister, both of whom had cavitated disease. In June, 1950, radiological examination showed bilateral disease with cavitation. The patient was admitted to a sanatorium, where he was given bed rest with streptomycin, 54 g., and P.A.S.; a right artificial pneumothorax was attempted and abandoned. In June, 1951, he was sent home. From December, 1951, to April, 1952, he was in the Brompton Hospital with a positive sputum. He was treated by postural retention for six weeks and then had a left phrenic crush with pneumoperitoneum; during this time he received streptomycin, 87 g., with P.A.S. He returned to a sanatorium for five months and was then readmitted to the Brompton Hospital on September 1, remaining there till November 5. His sputum was still positive on culture. Tomography revealed a small cavity at the right apex. On October 1 a right lucite plombage was performed, after which he was dyspnoeic for some days. Sputum disappeared soon after this, and he has been doing part-time work since December, 1953. Pneumoperitoneum treatment continues. The total dose of streptomycin received by this patient was 182 g.

Bacteriologists and morbid anatomists in this and other countries are investigating the finding in many resected specimens that large numbers of bacilli are present in some lesions and none in others; only a small proportion of these bacteria will grow on the artificial media now in common use, and may fail to infect or kill laboratory animals.

Dubos (1953), in his summing up of a discussion on the subject at the twelfth Conference on the Chemotherapy of Tuberculosis held in February, 1953, in the United States, comments on these facts. Bacilli are found more often in cavitated lesions than in closed necrotic foci; these bacilli may be dormant and not necessarily dead. A low-oxygen, high carbon-dioxide content is known to inhibit the growth of tubercle bacilli *in vitro*, and these are just the conditions found in closed necrotic lesions. Breakdown products of necrotic tissue such as spermine and spermidine, and enzymes such as lysozyme, also inhibit bacterial growth *in vitro*. Bacilli may, however, persist in the tissue-cells and lymph nodes of men and animals in spite of prolonged chemotherapy.

Further research is required both by laboratory workers and by clinicians who will make accurate observations on the anatomy, pathology, and bacteriology of these pulmonary lesions, follow up the patients, and publish their findings.

There is no doubt that in the right type of disease resection is a fundamental treatment which has come to stay—and rightly so. At present I am uneasy about the policy of some surgeons in removing apparently "benign" lesions on the supposition that these may break down at some future date.

When one ponders over the problem of pulmonary tuberculosis one is always brought back to the question of immunity. Why should the rat be immune and not the guinea-pig? Recently I have been studying sections of lung

taken at biopsy from patients with miliary shadows in the lungs. Some of these cases are diagnosed on all available evidence as "sarcoidosis," and others as benign chronic miliary tuberculosis; a small group seems to be "on the fence" between the two. I have a feeling that further experimental study of the syndrome called "sarcoidosis" may provide a clue to the problem of immunity in tuberculosis.

Summary

The treatment of pulmonary tuberculosis is reviewed in the light of recent advances.

The aims of treatment are: (1) permanent healing of the lesions; (2) minimal loss of respiratory function; and (3) the attainment of these in the shortest time.

In addition to the long-established principles of rest, diet, fresh air, and graded exercises there are three main therapeutic weapons: (1) antibacterial drugs without collapse therapy; (2) collapse therapy, either medical or surgical; and (3) resection of the diseased portion of lung.

The value of the various forms of treatment is assessed on the basis of results obtained by the author and by other workers in Great Britain.

The finding that large numbers of tubercle bacilli are present in some lesions and none in others is referred to, and the need for further research into this question is stressed.

Further experimental study of the syndrome called "sarcoidosis" may provide a clue to the problem of immunity in tuberculosis.

My grateful thanks are due to my registrars, Dr. J. K. Gurling and Dr. W. H. Helm, for their valuable assistance in reviewing the records of my cases; also to my secretary, Miss Hope Daniel, for her persevering efforts in following up the patients.

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In his Report for 1953, Dr. Stuart Laidlaw, Medical Officer of Health for the City of Glasgow, writes: "One can readily imagine the public outcry had diphtheria immunization been available only to the well-to-do. But it is little encouragement to those protagonists of free preventive measures that so few avail themselves of the opportunities offered. Public apathy is not restricted to diphtheria immunization alone. Only 17% take advantage of free infant vaccination, though Glasgow is a seaport town with an air terminal. Many contacts of open cases of tuberculosis decline to be *x*-rayed. Many parents ignore the opportunity of B.C.G. protection for children of school-leaving age. There is also the unsatisfactory response by expectant mothers to the offers of free dental treatment and supplementary vitamins despite the fact that these provide additional safeguards to themselves and the unborn child."

CURRENT THERAPY IN PULMONARY TUBERCULOSIS IN THE UNITED STATES*

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[WITH SPECIAL PLATE]

The study forming the subject of this lecture was carried out under the auspices of the Committee on Non-Surgical Collapse Therapy of the American College of Chest Physicians to determine the kind of therapy advised as the initial treatment in different cases of pulmonary tuberculosis by physicians who are recognized experts in this disease. Ten representative cases were selected. They were of ordinary forms of tuberculosis that might be seen any day in such a physician's office. They differed as regards age, race, sex, and type of disease, but had no unusual or bizarre complications. All had positive sputum. They could be grouped, according to the classification of the National Tuberculosis Association, into minimal, moderately advanced, and far advanced. These patients had been known to us for a long time. It was endeavoured to make the initial problem as simple as possible. A similar study was carried out on these same ten cases in 1948.

The method of investigation was as follows. A brief case history with a single chest radiograph of each case was sent, with an accompanying questionary, to the one hundred physicians selected as representative of the top clinical thinking in their respective areas throughout the United States. The questions asked were: (1) Would you use bed rest alone as therapy for this type of case? (2) Would you add drug therapy? (3) Would you use collapse therapy? (4) Would you use surgery? (5) With the therapy indicated above, how long would you keep this patient on complete bed rest and in a sanatorium? In general, complete bed rest is interpreted as strict rest in bed with no bathroom privileges; the patient is not allowed out of bed at all for any reason. Modified bed rest is indicated as such.

In what follows the replies to the above questionary are analysed.

Drug Therapy

Drug therapy will be discussed for all of the cases together, because the same practitioners recommend practically the same dosages throughout the ten cases and most of them say they would start chemotherapy immediately. The length of time chemotherapy would be continued varied considerably, however, for the different patients.

Antimicrobial therapy is now generally accepted by the vast majority of chest physicians as part of the routine treatment of pulmonary tuberculosis, whatever the extent of the disease. Such therapy would be used as part of the basic treatment by 97%, with variation, according to the extent of the disease, only from 93% for minimal disease to 99.5% for far advanced disease. The drugs of choice were limited to streptomycin, *para*-aminosalicylic acid (P.A.S.), and isonicotinic acid hydrazide (isoniazid). Practically all the physicians would use combined and continuous therapy, with a marked preference for streptomycin plus P.A.S. A majority favoured the intermittent use of streptomycin in a dosage of 1 g. twice weekly, with P.A.S. by mouth to tolerance—about

*Based on the Fourth Varrier-Jones Memorial Lecture, given at Manson House, London, on May 14, 1954.

J. L. LIVINGSTONE: OBSERVATIONS ON THE TREATMENT OF PULMONARY TUBERCULOSIS AT THE PRESENT TIME

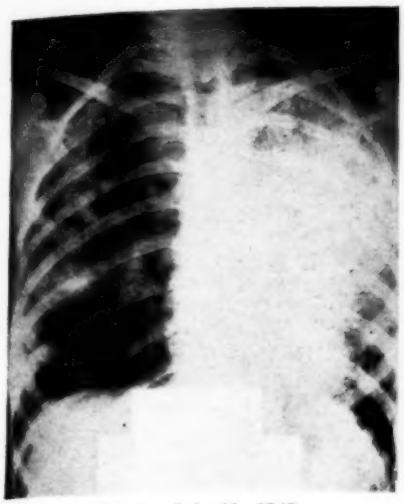


FIG. 1.—July 11, 1949.

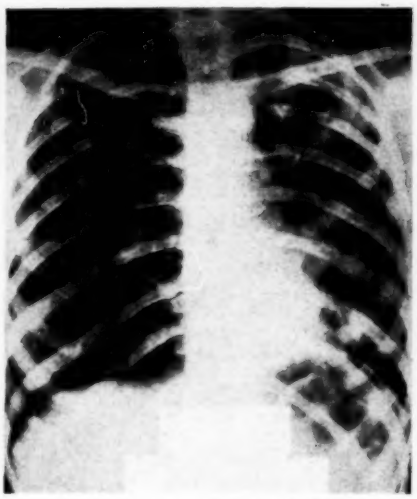


FIG. 2.—December 29, 1949.

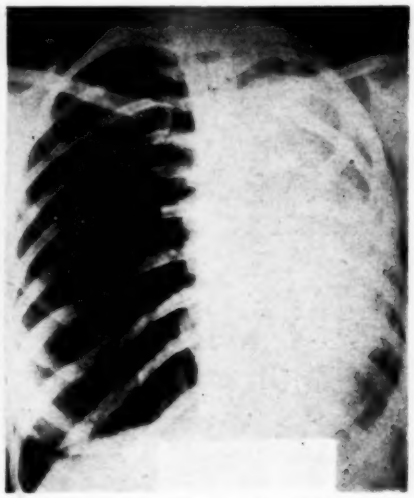


FIG. 3.—February 19, 1954.

FIGS. 1-3: Case A.

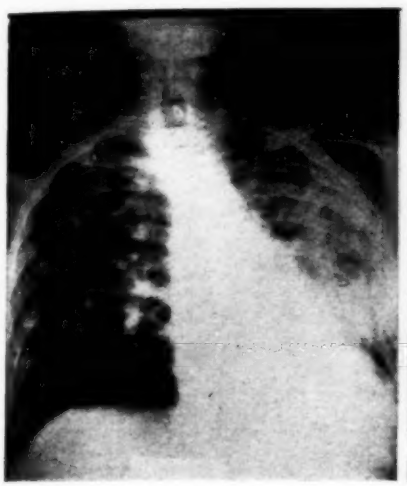


FIG. 4.—July 10, 1948.

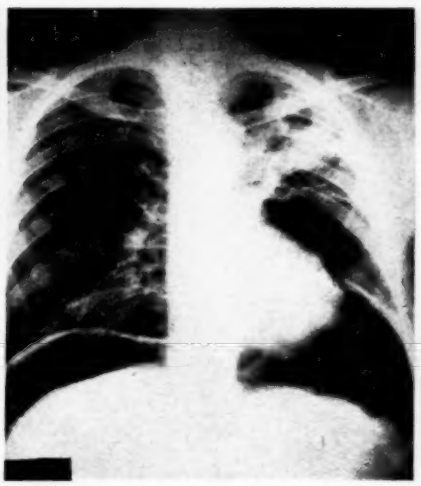


FIG. 5.—February 12, 1952.

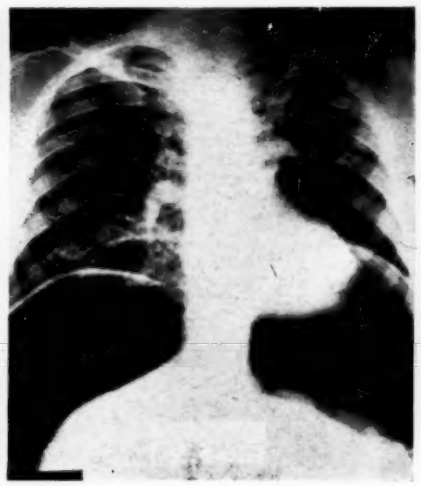


FIG. 6.—May 5, 1954.

FIGS. 4-6: Case C.

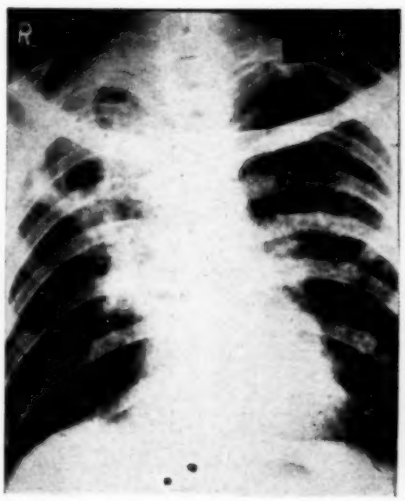


FIG. 7.—December 6, 1951.

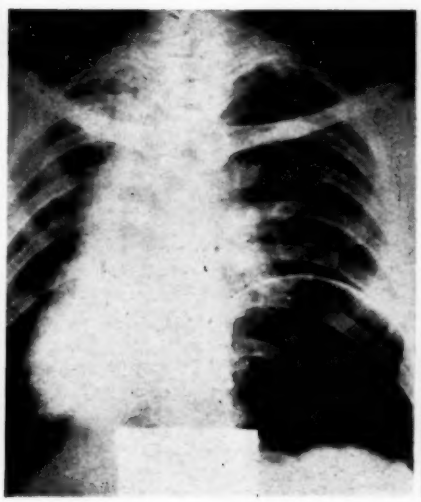


FIG. 8.—September 29, 1952.

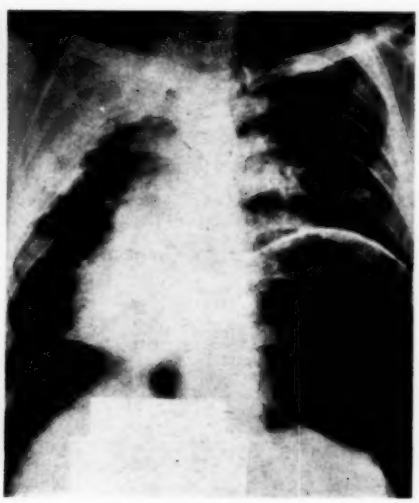


FIG. 9.—May 6, 1954.

FIGS. 7-9: Case D.

FIG. 1.—Case III: Minimal bilateral tuberculosis with right pleural effusion.

FIG. 2.—Case VIII: Moderately advanced tuberculosis with solitary 3-cm. cavity at right apex.

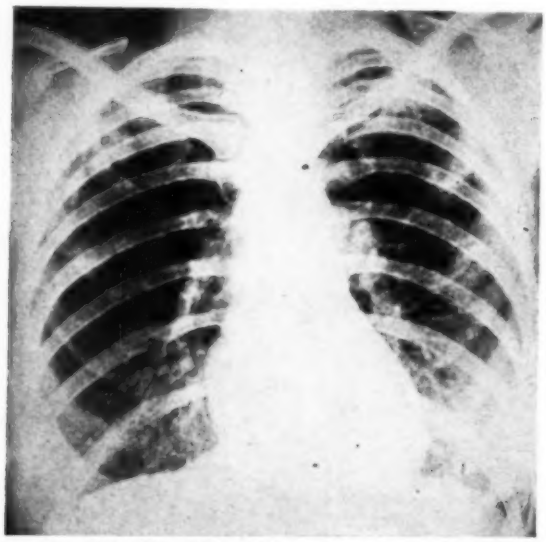


FIG. 3.—Case IX: Moderately advanced acute bilateral tuberculosis, more extensive on left.

FIG. 4.—Case II: Far advanced tuberculous pneumonia.

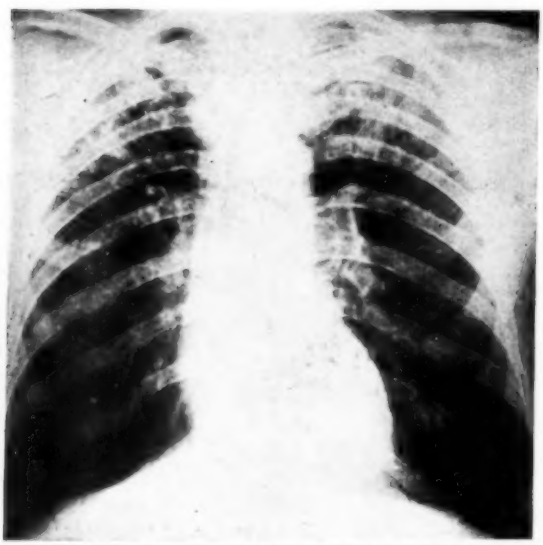


FIG. 5.—Case X: Far advanced bilateral fibrocaseous lesions with cavity on left.

A. CHAKRAVARTI and G. CHAKRAVARTI: GIANT-CELL POLYPHLEBITIS

Section of vein showing thickened and infiltrated wall with collections of giant cells (arrowed). ×65.

12 g. daily. There was less agreement about the dosage of isoniazid, which varied from 3 mg. per kg. of body weight to 10 mg. per kg. with a majority using 3 to 5 mg. per kg. In the more acute far advanced cases there was a definite leaning toward combining all three drugs.

In the United States there is a strong trend toward standardization in the use of antimicrobial therapy. This is probably greatly influenced by what is called the Annual Conference on Chemotherapy of Tuberculosis under the auspices of our Veterans Administration, which acts as a forum for the presentation of various studies on the subject.

When the views on the length of time such therapy should be continued are studied no pattern evolves. There were recommendations for courses of from one month all the way to continuous therapy for three years. In minimal and moderately advanced disease a majority thought in terms of one year or less, with a considerable number favouring less than eight months; while in far advanced disease a majority would continue giving the drugs for one to two years. However, in both instances there were many recommendations that these broad generalizations would not cover.

As a corollary to the above it is interesting to note that there are now very few who use bed rest alone in the treatment of pulmonary tuberculosis—only 1%, in fact, of the total reporting. However, when we consider the use of antimicrobial therapy plus bed rest we find that 37% of the group as a whole favoured this as the treatment of choice. This percentage was made up very largely from the answers on the treatment of minimal disease, in which nearly three-quarters of the group favoured antimicrobial therapy plus bed rest.

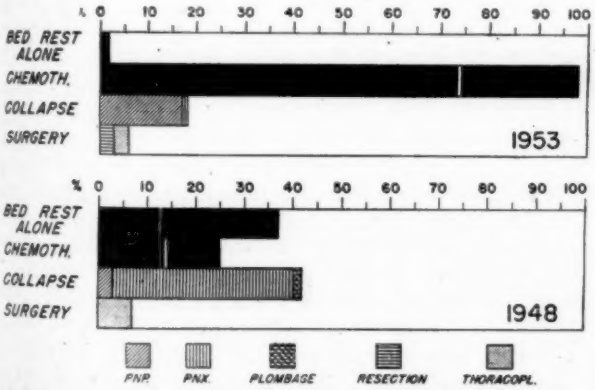
Since 1948 the use of bed rest plus drugs and without collapse therapy has tremendously increased, at the expense of collapse therapy.

Treatment Recommended in Five Sample Cases

I will not discuss each of the ten cases separately, but will use as examples one minimal, two moderately advanced, and two far advanced cases (minimal, Case III; moderately advanced, Cases VIII and IX; and far advanced, Cases II and X).

Case III: Minimal pulmonary tuberculosis, bilateral, productive, with pleural effusion on the right, in a 43-year-old white female. Chest radiograph (Plate, Fig. 1) revealed a homogeneous density in the right pleural cavity (pleural effusion) and infiltration at both apices—more marked on the right.

The graph for this case (Graph 1) shows that in 1953 practically no one would treat this pleural effusion in association with minimal bilateral tuberculosis by bed rest alone, in contrast to 37% in 1948. However, approximately three-fourths would use bed rest plus chemotherapy. As many as 98% would use chemotherapy plus other methods, with a marked decrease in the use of collapse therapy since 1948, largely at the expense of pneumothorax, which fell from 37% to 1%. A very few would think surgery advisable for this patient.

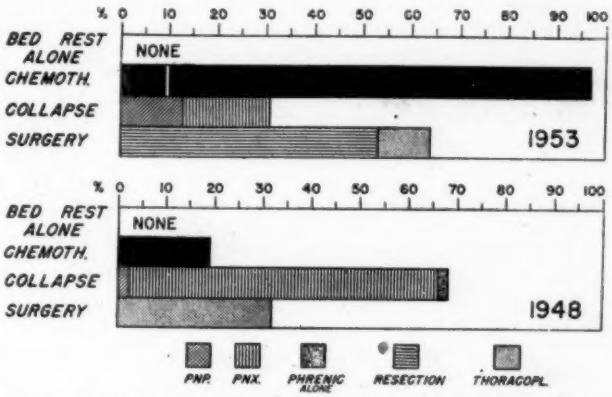


GRAPH 1.—Case III: Comparison of recommendations for treatment in 1953 with those in 1948. Chemotherapy alone with bed rest was prescribed by 74% in 1953, as against 14% in 1948 (indicated by vertical dividing line).

Case VIII: Moderately advanced pulmonary tuberculosis, unilateral, with a solitary 3-cm. cavity at the right apex, in a 34-year-old white male (Plate, Fig. 2).

Most physicians would now use chemotherapy. One-third would initiate a prompt reversible collapse, about half of these using pneumoperitoneum and the other half a right pneumothorax. Two-thirds would, however, prefer surgery after the lesion had shown some stabilization by bed rest plus drugs. The large majority would expect maximum response to drug treatment and relatively stabilized lesions within three to six months. Fifty-two physicians would prefer resection and eleven a thoracoplasty.

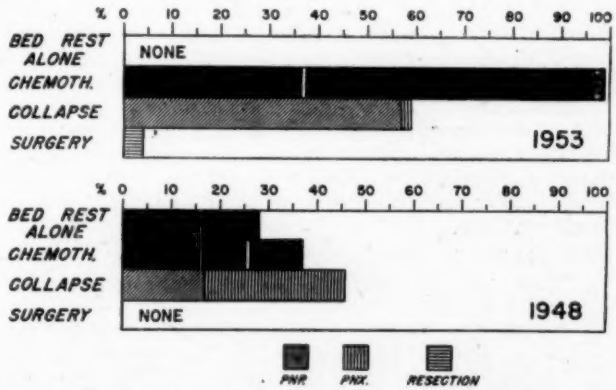
If the treatment prescribed now is compared with that recommended in 1948 (Graph 2) it will be seen that in the case of this 34-year-old man with moderately advanced disease and a unilateral cavity nearly all would use chemotherapy, with a definite reduction in the use of reversible collapse therapy from 68% in 1948 to 31% at the present time. It is noteworthy that there has been a marked drop in the use of pneumothorax in favour of pneumoperitoneum since 1948. It is interesting, however, that 18% would still try pneumothorax, and continue it for three to five years in most instances if the collapse promptly became adequate. But surgery was the treatment of choice for this patient.



GRAPH 2.—Case VIII: Comparison of 1953 with 1948. Chemotherapy alone with bed rest prescribed by 10% in 1953 and by none in 1948.

Case IX: Moderately advanced pulmonary tuberculosis, acute, bilateral, more extensive on the left, in a 34-year-old white female. Chest radiograph (Plate, Fig. 3) revealed a soft mottled infiltration in both upper lung fields, more extensive on the left.

Practically all the physicians would use chemotherapy in this case, and six out of every ten would favour reversible



GRAPH 3.—Case IX: Comparison of 1953 with 1948. Chemotherapy alone with bed rest prescribed by 37% in 1953, as against 26% in 1948.

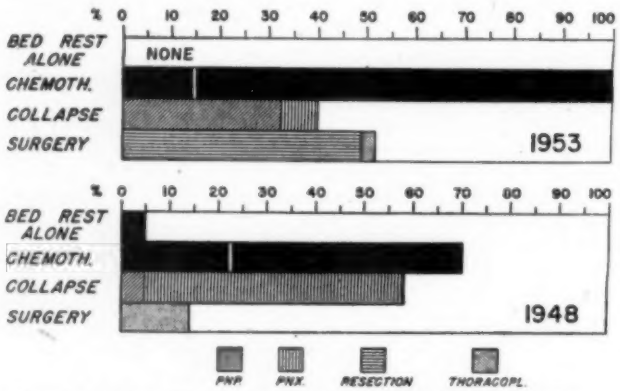
collapse, only a very few advising resection. The collapse therapy recommended was pneumoperitoneum. No one at the present time would treat this moderately advanced, acute bilateral disease in a 34-year-old woman with bed rest alone, as compared with 28% in 1948 (Graph 3). However, 37% would use only bed rest plus chemotherapy. There has been some increase since 1948 in the number who would use reversible collapse therapy, 57% favouring pneumoperitoneum, while only 2% would use pneumothorax, in contrast to the 29% who recommended pneumothorax in 1948. Four would now advise surgery as the primary procedure, advocating resection.

Case II: Far advanced tuberculous pneumonia in a 31-year-old white female. Chest radiograph (Plate, Fig. 4) showed a diffuse homogeneous density occupying the upper half of the right lung field and containing a cavity.

All the physicians would use drugs in this case, 42% prescribing streptomycin and P.A.S., 30% streptomycin, P.A.S., and isoniazid, while 28% would use a combination of any two of these drugs. Thirty-two physicians would use pneumoperitoneum and eight would try to establish a right pneumothorax. Forty-nine would expect to do a resection after a period of bed rest and drug treatment lasting from five to ten months. Three would prefer thoracoplasty.

In this far advanced case of tuberculous pneumonia in a 31-year-old woman no one would treat the patient with bed rest alone, and there was complete agreement as to the use of combined antimicrobial drug therapy. There was a tendency to use all three drugs in this case and to give streptomycin daily at the beginning. The majority would use the drugs for from twelve to twenty-four months.

The use of collapse therapy has fallen from 58% in 1948 to 40% now (Graph 4), largely at the expense of pneumothorax, with a correspondingly large increase in the use of

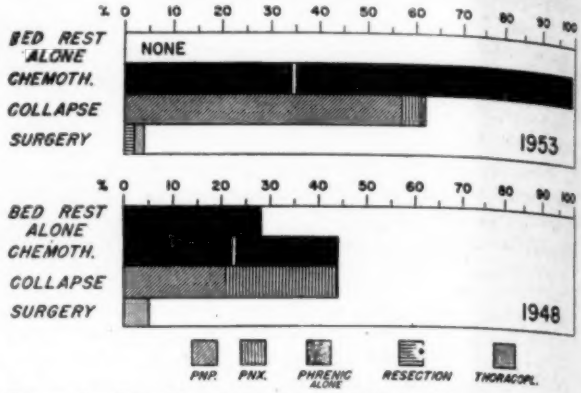


GRAPH 4.—Case II: Comparison of 1953 with 1948. Chemotherapy alone with bed rest prescribed by 15% in 1953, as against 23% in 1948.

pneumoperitoneum. Three years seemed to be the recognized time for pneumothorax to be continued. There was much disagreement as to how long to continue pneumoperitoneum, the largest group favouring five years, but substantial numbers indicated three or four years and a few suggested shorter periods. Note the definite increase in the use of resection at the expense of thoracoplasty and collapse therapy. Physicians who favoured resection tended to recommend a longer stay in a sanatorium than those who preferred collapse therapy.

Case X: Far advanced pulmonary tuberculosis, fibrocaseous lesions in the upper half of both lungs, with cavity on the left, in a 57-year-old white male. Chest radiograph (Plate, Fig. 5) showed old calcified lesions at the right apex, multiple scattered nodules, little fibrosis, but soft infiltration throughout the right lung in the upper two-thirds. In the left lung there was a soft mottling throughout the upper third, with a 1.5-cm. cavity in the first interspace.

In this case of far advanced pulmonary tuberculosis with cavitation and bilateral fibrocaseous disease in a 57-year-old man no one would now use bed rest alone, but 35% would use only bed rest plus chemotherapy. More would use reversible collapse therapy now than in 1948—almost two-thirds, compared with 44% in 1948 (Graph 5). There is a marked shift toward the use of pneumoperitoneum. All would now use chemotherapy. Very few considered this a case for surgery.



GRAPH 5.—Case X: Comparison of 1953 with 1948. Chemotherapy alone with bed rest prescribed by 35% in 1953, as against 23% in 1948.

Summary of Recommendations

The recommendations for treatment can best be summarized by grouping the ten cases according to the three degrees of severity referred to earlier.

For the minimal group few would use bed rest alone. 72% would use bed rest plus chemotherapy. About 20% more would add various forms of collapse, mostly pneumoperitoneum, but a few pneumothorax. Even in the minimal cases a small percentage would recommend surgery, mostly resection.

In moderately advanced disease a very few would recommend bed rest alone. Less than 40% would use chemotherapy alone with bed rest, a larger number—60%—adding collapse or surgery. Three-fourths of those advising collapse would use pneumoperitoneum, the other one-fourth pneumothorax. Resection was recommended by 20%.

In far advanced disease no one recommended bed rest alone. A small number would use chemotherapy plus bed rest. About 80% recommended collapse therapy or surgery with chemotherapy and bed rest. Almost 60% would use pneumoperitoneum; 21% would perform resection.

Concluding Remarks

Just a personal word in closing. We have found this study very interesting. We actually treated these patients somewhat differently. Our own therapy would be complete bed rest until the sputum disappeared or became negative on examination by the concentration method. All of these patients would receive drugs, usually a combination of all three and for a period of approximately eighteen months. We use much reversible collapse therapy, with pneumoperitoneum as the method of choice and as the initial treatment in most cases. We review our results in three months, and if cavities that are not well on the way to closing are present we may then consider pneumothorax. However, all patients are examined bronchoscopically before being subjected to this procedure. Actual tracheo-bronchial disease is a contraindication to pneumothorax, though it is not to pneumoperitoneum. We may then turn to surgical procedures. With some patients we feel from the beginning that surgery will eventually be necessary. We use bed rest plus drugs plus pneumoperitoneum as a

damping-down method for the acute exudative lesions, expecting the surgeon to remove the cavity-bearing area at a later date.

Tracheo-bronchial disease is still a problem. Many patients in whom tuberculous disease of the walls of the bronchi can be visualized through the bronchoscope are not permanently cured by drugs. If such tracheo-bronchial disease is removable, we feel that localized segmental resection is eventually indicated.

We have termed these methods a "dynamic approach" to the problem of tuberculosis. With prompt diagnosis and adequate treatment more patients with pulmonary tuberculosis are now getting well than ever could have done so previously.

Summary

The trend of treatment of pulmonary tuberculosis in the United States is discussed on the basis of a questionary sent in 1953 to one hundred physicians in that country. Recommendations were sought as to the treatment of ten selected cases of the disease. The replies are analysed and compared with those received in answer to a similar inquiry in 1948 concerning the same ten cases.

The investigation has shown that since 1948 the use of bed rest plus drugs and without collapse therapy has enormously increased. Collapse therapy is, however, still favoured in cases of more advanced disease, but pneumoperitoneum seems to have largely replaced pneumothorax as the procedure of choice.

A CASE OF GIANT-CELL POLYPHLEBITIS

BY

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AND

GANEN CHAKRAVARTI, M.B.

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[WITH SPECIAL PLATE]

Since 1941 attempts have been made to bring under a single heading such apparently diverse clinical conditions as polyarteritis nodosa, lupus erythematosus, scleroderma, and dermatomyositis. Banks (1941) has suggested that there is a common denominator in these diseases. Klemperer, Pollack, and Baehr (1942) proposed grouping them under the term "collagen diseases," fibrinoid degeneration of collagen having been found to form the basis of all of them. Claims have also been advanced for including in the group such diseases as rheumatic fever, Buerger's disease, and even serum sickness and the calcinoses. In spite of attempts at generalization, however, the "collagen group" of diseases remains an assortment of heterogeneous clinical varieties, and the specific conditions which should be included or excluded continue to be a matter for speculation.

Polyarteritis nodosa is predominantly a vascular collagen disease. Recognized as early as 1866 (Kussmaul and Maier), it has received widespread recognition only in recent years. Though an appreciable number of cases have been reported in the world literature, so far only three cases occurring in India are on record, the first of which was reported by one of us (Chakravarti, 1951). It must be appreciated, however, that polyarteritis nodosa has hitherto been recognized as an entity only on the basis of the clinical findings; that it has any relationship to infection, hypersensitivity, or allergy is far from being established, and experimental attempts to arrive at its aetiology have been without success. But, unless the aetiology of such diseases as this is established beyond doubt, cases of arteritis presenting aberrant manifestations differing from the recognized clinical picture will raise problems of identification and lead to claims for the discovery of new entities. Thus a variety of arteritis with a different age incidence, vascular distribution, and prognosis was described in 1932 by Horton, Magath, and Brown. They gave it the name of "temporal arteritis." Giant cells formed the distinctive feature in its pathology, and, though it was claimed that giant-cell arteritis and polyarteritis are identical, this view was not supported by Harrison (1948).

In the light of the above considerations we describe below a case in which the clinical picture resembled in some respects that of polyarteritis nodosa, yet differed from it in a number of essential features. Clinically it was considered to be an example of polyphlebitis rather than of polyarteritis, but histological examination of the vascular lesions revealed the presence of giant cells, so long considered almost characteristic of temporal arteritis. The case would therefore appear to be of a hitherto unrecognized type of arteritis.

Case Report

A married woman aged 28 years; mother of four children, the last of which was born six years ago. No history of miscarriage. Past history of colitis, lasting three months, seven years ago. Streaks of blood in sputum during a febrile bout lasting one week three years ago. Husband's two brothers died of pulmonary tuberculosis, six and three years ago respectively.

Present Illness

The patient had suffered from a low-grade, irregular fever and malaise for the past year. One year previously slightly painful, tender spots had appeared on the medial aspects of the thighs, followed by a similar spot on the anterior aspect of the right forearm. For six months there had been persistent and gradually progressive anorexia and asthenia. A constant dull pain in the left hypochondrium, with exacerbations when the patient lay on her left side and on walking, had been noticed for the past three weeks. Since the last attack of colitis seven years ago there had been frequent bouts of diarrhoea. There were no cardiac complaints, no subjective urinary symptoms or respiratory trouble, and no signs of mental disturbance.

Clinical Examination

On examination the patient was found to be sickly, of asthenic build, pale, and anaemic. In the left eye there was phlyctenular conjunctivitis. Temperature 99° F. (37.2° C.); pulse rate 128 and respirations 32 per minute. The fingers were not clubbed. There was no enlargement of cervical or axillary lymph nodes, but the inguinal nodes showed discrete enlargement; these were neither painful nor tender. The spleen was palpable three fingerbreadths below the costal margin; it was soft and acutely tender, but there was no evidence of perisplenitis. The liver was palpable two fingerbreadths below the costal arch; it was rather soft, but peritoneal or paravertebral tenderness was absent. No abnormality was detectable in the colonic or renal area or in the lungs. There was no obvious dilatation of the heart; a harsh systolic murmur was audible all over the praecordium, with maximum intensity in the left third space, in the parasternal line, conducted well out to the posterior axillary line, but no thrill or associated diastolic murmur; the pulmonary second sound was accentuated. Blood pressure 108/64 mm. Hg.

On the medial side of the right arm, just above the elbow-joint, a thick subcutaneous tender lump containing a non-pulsatile cord-like structure could be felt. Similar non-pulsatile cord-like structures containing tender nodules were present on the front of the right forearm and the medial aspects of the thighs; these were the painful, tender spots complained of by the patient. A nodule was also present in the course of the cephalic vein on the right arm. The superficial arteries felt normal and had pulsations in them, except the left radial and brachial arteries, which could not be felt. This was probably a congenital abnormality with adequate compensation by way of collateral channels, since the nutrition of the limb was well maintained. There was no subjective or objective evidence of peripheral vascular disturbance.

Laboratory Investigations

Blood: R.B.C., 3,750,000 per c.mm.; Hb, 60%; W.B.C., 11,000 per c.mm. (polymorphs 75%, lymphocytes 18%, monocytes 2%, eosinophils 5%); E.S.R., 144 mm. in 1 hour (Westergren); coagulation time, 2 minutes; bleeding time, 3½ minutes; blood culture (repeated after two weeks), no growth; blood urea, 24 mg. per 100 ml.; Wassermann reaction negative. Urine: albumin, trace; pus cells, few; red blood cells, occasional; culture, *Alcaligenes faecalis*; no acid-fast bacilli seen by concentration method (examined thrice at intervals of two weeks). Stool: few degenerated pus cells; occasional red blood cells; no protozoa; no acid-fast bacilli seen by concentration method (examined twice at intervals of two weeks). Sputum: no acid-fast bacilli demonstrated by concentration method (examined twice at intervals of two weeks). Peripheral blood tested for "L.E. cells" found to be negative. Tuberculin test just positive with 1 in 1,000 dilution.

Special Investigations

Skiagrams of chest (taken at intervals of two months) showed no evidence of any parenchymatous lung infiltration. No evidence of calcified lymph nodes could be seen in a flat radiograph of the abdomen. Radiological examination of bones and joints distal to the affected vessel in the forearm showed no detectable abnormality. Ophthalmoscopic appearances were normal. An electrocardiogram gave evidence of sinus tachycardia with a normal sinus rhythm and normal auriculo-ventricular conduction; there was no evidence of coronary disease or of ventricular enlargement.

Histological Examination

Subcutaneous Forearm Vein.—The vein was thickened, cord-like, and thrombosed. On section the outer coat showed haemorrhage and many new, thin-walled blood vessels throughout its thickness. In some areas there was fibrinoid degeneration, patchy in distribution. Infiltration by round cells, of which the principal cells were lymphocytes together with a few plasma cells and monocytes, was of almost uniform distribution. At the junction of the outer and middle coats there were multinucleate giant cells surrounding the whole circumference like a ring. The middle coat showed interruption by granulation tissue. The muscle fibres were separated from one another by vascular connective tissue and lymphocytes. Minute patchy areas of degeneration were also seen in the middle coat. The intima was thickened by cellular connective tissue, in which a few capillaries were also seen. The lumen was greatly narrowed, but no thrombus was present, and the appearance was suggestive of organization and recanalization of a previous thrombus. (See Special Plate.)

Cephalic Vein.—A small area of thickening was felt, but the vein appeared otherwise normal. On section the outer coat showed a few areas of focal collections of lymphocytes in which could be seen a few multinucleate giant cells; there were also small areas of haemorrhage and degeneration. In the middle coat the findings were the same as in the specimen from the forearm. The inner coat was more or less normal.

Inguinal Lymph Node.—There were patchy areas of fibrinoid degeneration, but no giant cells were seen. The follicular structure was unaltered and the capsule intact.

A small arteriole from the right forearm adjacent to the affected vein also showed no abnormality.

Discussion

In arriving at a diagnosis in this case there would, in the first place, seem to be little difficulty in excluding other known forms of acute phlebitis. Thrombophlebitis migrans, for example, is of short duration and is characterized by a polymorphonuclear leucocytosis. "Tropical primary phlebitis," which was described by Fisher, Fisher, and Lendrum in 1947, runs a self-limiting course, and recurrence is uncommon. Histologically the granulation tissue interrupting the vessel wall is composed of new capillaries, polymorphs, and polyblasts, some of the last-named containing phloxinophil inclusion bodies. Again, in thromboangiitis obliterans there may be an associated phlebitis, but the disease is afebrile and characterized by arterial obstruction in the limbs; moreover, giant cells are found only in the region of organizing blood-clot. Secondly, although the family history, together with the clinical course, might suggest chronic miliary tuberculosis, absence of positive evidence of tuberculosis and the fact that the lesions were limited to the systemic veins would rule out such a possibility.

It seems certain that the case belongs to the group of vascular collagen diseases. Of these, the conditions that seem worth considering in the differential diagnosis are: (1) lupus erythematosus, (2) giant-cell arteritis, and (3) polyarteritis nodosa. (1) In acute disseminated lupus erythematosus skin lesions may be absent, but, in addition to endocarditis, involvement of serous membranes and joints is an important feature. Moreover, this is rather a visceral involvement than a surface vascular manifestation. Clinical diagnosis is dependent on the demonstration of L.E. cells. (2) Giant-cell arteritis usually affects the aged, the lesions being chiefly confined to the scalp and cranial arteries; the veins are never affected. (3) Polyarteritis nodosa is essentially a condition of primary arteritis usually affecting males. Hypertension and kidney involvement form an essential part of the clinical picture, and leucocytosis is common. Histologically, giant cells are seldom encountered.

Our case, therefore, though most closely resembling polyarteritis nodosa in the widespread distribution of the vascular lesions, has definite points of difference. Thus there was no hypertension or renal failure even twelve months after the onset of the illness; leucocytosis was absent; lesions were limited to widely scattered veins; and, finally, giant cells were present. These would appear to be grounds for regarding the case as at least a variant form of the collagen group of diseases which has not hitherto been described.

The histological slides were sent to Professor J. H. Dible, of the Department of Pathology, Postgraduate Medical School of London, for his opinion, and he makes the following comments: "I have examined the tissues and have also discussed them with Dr. Harrison. He points out that giant-cell arteritis, so far as is known, never affects veins, and, moreover, it is essentially a disease of advanced years. I don't think either the history or the histology of the lesion fits in with syphilis. . . . I have never seen tuberculosis attack veins in this way. . . . The disease which seems most worth considering is polyarteritis nodosa. The limiting of lesions to the veins and the peculiar picture of giant cells are certainly unusual, but, then, so is the whole case. I don't think this disease, either clinically or histologically, is the same as those reported by Fisher, Fisher, and Lendrum (1947)."

Summary

A case is reported of a protracted febrile illness in a young married woman in which polyphlebitis, inguinal lymphadenopathy, disproportionate tachycardia, and a high erythrocyte sedimentation rate were notable

features. The condition in many respects resembled polyarteritis nodosa, but differed from it in that the vascular lesions were limited to the veins and in the presence of giant cells.

A full account of the clinical and laboratory findings is given and the differential diagnosis discussed.

The patient is alive and is being followed up.

We are most grateful to Professor Dible for his valuable opinion on the tissue slides. Our thanks are also due to Dr. C. V. Harrison, Reader in Morbid Anatomy, University of London, for his views on giant-cell arteritis, and to Drs. S. N. De and A. Mukherjee, of the Departments of Pathology at the Sir Nilratan Sarkar Medical College and School of Tropical Medicine, Calcutta, respectively, for their help and co-operation.

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SPONTANEOUS MYXOEDEMA DEVELOPING AFTER THYROTOXICOSIS

BY

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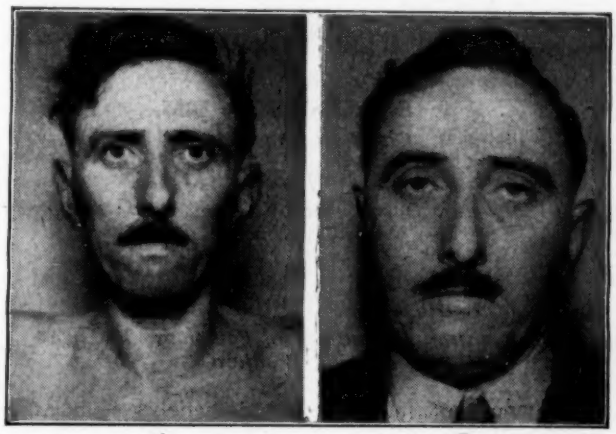
Primary adult myxoedema most commonly occurs from idiopathic primary atrophy, but may also follow thyroidectomy, excessive radio-iodine dosage, or destructive thyroid disease such as lymphadenoid goitre (Hashimoto's thyroiditis). It is also frequently stated in textbooks that adult myxoedema may arise in patients who have previously had frank thyrotoxicosis ("burned out toxic goitre"), but it is exceedingly hard to discover records of such cases. Crotti (1938) describes one, but gives the impression that he has seen many more: he intimates that the progression from hyperthyroidism to hypothyroidism takes many years. McGavack (1951) describes the case of a patient who received only iodine treatment for his thyrotoxicosis but developed myxoedema some four years later. In view of the paucity of cases recorded, we are reporting one which was first treated for thyrotoxicosis with antithyroid drugs for 21 months; an active thyroid gland was still present at the end of this time. Twelve months later the patient was definitely myxoedematous.

Case Report

A married man aged 40 first presented in November, 1950, with an 18-months history of increasing fatigue, intolerance of hot weather, and 3 st. (19 kg.) loss of weight. He also complained of some dyspnoea on exertion and of some prominence of the eyes in the past two months. His appetite was very good. On examination at that time he was a thin, flushed, and anxious man (Fig. 1 A) with a fine tremor of the fingers, an increased peripheral circulation, and a slightly enlarged soft thyroid gland with a bruit. There was no true exophthalmos, lid retraction, or lid lag, but a definite "stare." There were no other physical signs. Investigation confirmed the diagnosis of hyperthyroidism (Fig. 2). The basal metabolic rate ranged from 140 to 160% of normal according to the Boothby standards (52 to 60 cals./square m./hr.). A radio-iodine test yielded only 10.7% excretion in 48 hours ("T" index of thyroid uptake = 105 compared with the normal of 3–13) (see Fraser *et al.*, 1953).

After a short course of an anti-thyroxine drug (butyl-4-hydroxy-3:5-diiodobenzoate) (Fraser and Maclagan, 1953), and after an episode of pyrexia which was found to be due to otitis media, the patient was discharged on methylthiouracil, 600 mg., and thyroid, 1 gr. (65 mg.), daily. He responded rapidly to treatment, gained weight, and remained well controlled on diminishing doses of thiouracil from January, 1951, to October, 1952. Antithyroid drug treatment was then stopped, and a radio-iodine test done a few days later showed that the gland was still active in that the 48-hour excretion was 16.7% (T=62). He was then given a short course of potassium iodide and retested in February, 1953, when the 48-hour radio-iodine excretion had risen to 77% (T=2.6). This was attributed to excessive ingestion of iodides as the patient appeared



A B

FIG. 1.—Photographs taken in November, 1950 (A), and in January, 1954 (B).

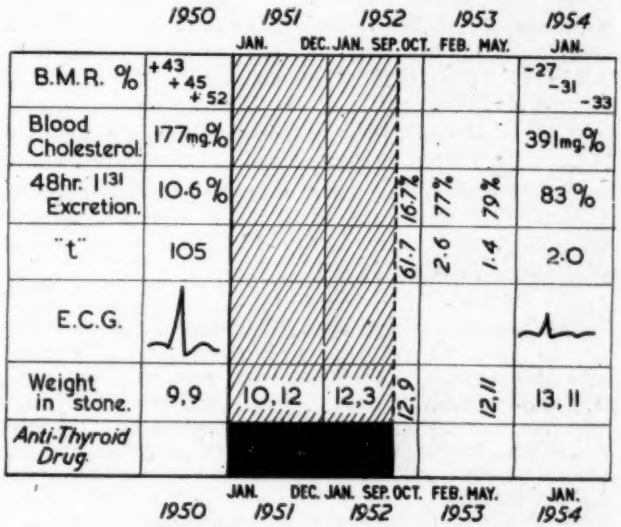


FIG. 2.—Results of investigation.

euthyroid, but when the test was repeated three months later it yielded a similar result, and when he was seen again in October, 1953, he had all the features of myxoedema—full puffy facies, husky voice, and dry skin—and his weight had risen to 13 st. (82.5 kg.) (Fig. 1 B). On admission for reinvestigation it was found that his blood cholesterol had risen to 391 mg. per 100 ml., his basal metabolic rate had fallen to −28 to −33% (25–28 cals./square m./hr.), his protein-bound iodine was 2.8 μg. per 100 ml., and he had

electrocardiographic evidence of myxoedema. There was no evidence of pituitary failure: he had a normal 17-ketosteroid excretion, and a normal diuresis in response to a standard water load.

Conclusion

Our data indicate that this patient had Graves's disease in 1950; that his thyroid gland was still functioning at the end of 1952, after treatment with thiouracil; and yet that one year later he had advanced primary myxoedema, which has since responded to thyroxine. We have been unable to find a record of any other case of myxoedema developing spontaneously so soon after thyrotoxicosis.

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SÉZARY'S SYNDROME

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Disturbances of the reticulo-endothelial system associated with generalized erythrodermia are well known, and form a diverse group, from which Sézary and Bouvrain (1938) first described a distinct syndrome. This is characterized clinically by bizarre features, and pathologically by the appearance of peculiar monocytoid cells in the blood. Sézary (1949) reviewed all the four published cases (Sézary and Bouvrain, 1938; Sézary, Horowitz, and Maschas, 1938; Baccaredda, 1939; and Sézary and Bolgert, 1942), to which Wilson and Fielding (1953) have added one more—the first reported in Britain.

The purpose of this paper is to draw attention to this syndrome by recording the clinical and pathological features of two further cases, and to discuss their relation to the reticuloses.

Case 1

The patient, a widow aged 77, previously led an active, healthy life. She had had smallpox at the age of 11 years and pneumonia at 73. Seven sons were alive and well; one premature infant was stillborn. About eight years ago she first suffered from attacks of extensive erythrodermia "just like measles," with varying degrees of exfoliation, and intense pruritus, starting in the feet and spreading over the limbs and trunk. These attacks were transient initially and were preceded by malaise. Later all her skin assumed a mottled appearance. Her palms were occasionally affected so severely that she was unable to do her housework. The mucous membranes were not affected, nor was there any history of excessive sweating, although she felt the cold intensely.

Some twelve months ago she became aware of "hot aches" and stiffness of the fingers, with distortion and looseness of the nails—so much so that she was unable to relieve the intense pruritus by "having a good scratch." Her skin also showed scattered patches of both leucodermia and brown pigmentation. Alopecia was complete apart from occasional tufts on the scalp. Recently oedema of the legs occurred, and infected excoriations have resulted in whitlows, and superficial ulcers on the ankles and elbows.

When first seen in 1950 she showed extensive erythema, fine desquamation, telangiectasia, and superficial lymphadenopathy. Various diagnoses were then considered, including parakeratosis variegata, pityriasis rubra pilaris, poikilodermia vasculare atrophicans, and lymphosarcoma. Skin and blood examinations at that time were equivocal. The presence of atypical white cells in the blood was noted, but their significance was not realized.

Clinical examination showed an elderly woman whose bizarre appearance is well illustrated by Fig. 1. The skin showed a generalized mottled erythrodermia with fine scaling and interspersed areas of slaty-brown pigmentation and leucodermia of varying sizes and indefinite distribution. Petechiae were maximal over the sternum and shins, the latter also showing erythema ab igne. The hair was absent apart from residual tufts on the scalp and a few remaining hairs on the pubes. The eyelids showed chronic blepharitis and ectropion of the lower lids. The nose was shrunken and the nares were widened. The joints of the wrists and knees showed arthritic changes. There were oedema, ulnar deviation, and flexural contractures of the digits of both hands: full extension, active or passive, of the fingers of the right hand was impossible. The nails and terminal phalanges of the left hand were almost entirely absent; the thumb was normal. The changes were less marked in the right hand, although all fingers showed some degree of deformity. The lymph nodes in the axillae and groins were all enlarged, firm, rubbery, and discrete. There was one similar lymph node in the right cervical area. The mucous membranes were normal.

FIG. 1.—Face and hands of Case 1.

No significant lesion has been revealed in any other system. The spleen was not felt. The liver edge was palpable just below the costal margin. There was slight oedema of the legs. B.P. 170/80. Weight 7 st. 12 lb. (49.9 kg.). The temperature and pulse were normal. *X*-ray films of the skull, spine, pelvis, and chest were essentially normal. The mental condition was that of a well-adjusted euphoric elderly woman.

Case 2

The patient, a married woman aged 40, had suffered from eczema as a child. Her appendix had been removed at the

age of 17 years. In 1948 she developed a patchy eczema whilst in hospital for recurrent erysipelas of the face. She has one daughter, aged 19 years.

About four years previously she first complained of pruritus starting on the trunk, followed by erythema of the arms, thickening of the skin, and generalized scaling. This started six months after laparotomy, at which the left tube and ovary—with a benign necrotic fibroma—were removed. "Drenching" night sweats followed by rigors were frequent. They ceased six months ago, since when she has felt the cold intensely.

During 1952 superficial lymphadenopathy occurred whilst she was under treatment elsewhere for generalized exfoliation. A provisional diagnosis of lipomelanic reticulosis was made and A.C.T.H. therapy instituted. Considerable improvement ensued, but A.C.T.H. was stopped after pigmentation and staphylococcal pyodermia supervened.

In January, 1953, she was admitted to hospital under our care with the same provisional diagnosis, the clinical features being those of intense pruritus, generalized erythrodermia, mild desquamation, pigmentation, and superficial lymph adenopathy. She continued to develop occasional pyodermic nodules, which have healed normally, although some required drainage.

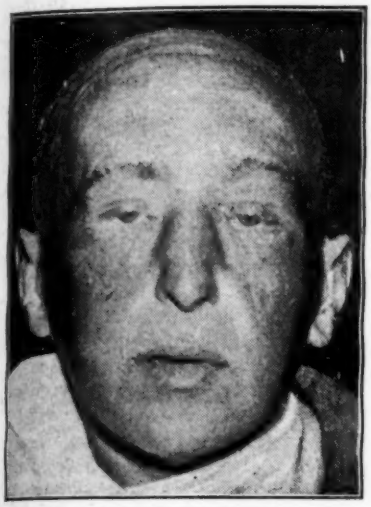


FIG. 2.—Face of Case 2.

Clinical examination showed a pathetic woman, whose appearance is shown in Fig. 2.

The skin showed generalized erythrodermia, fine scaling, and "mottling," which was more confluent on the lower parts. Generalized brown pigmentation was maximal on the face and trunk. Leucodermia was absent. The arms were lichenified and and there was some eczematization of the lower legs. Two right linear abdominal scars were present. At the time of writing there was one small pyodermic nodule on the right wrist. The hair was absent. The eyelids were normal, but the lower eyelashes were absent. The nose, joints, nails, and mucous membranes were normal. The lymph nodes in the neck, axillae, and groins were all enlarged, firm, rubbery, and discrete.

No significant lesion has been revealed in any other system. The spleen was not felt. The liver was palpable two fingerbreadths below the costal margin. There was slight oedema of the feet. B.P. 110/65. Weight 6 st. 5 lb. (40.4 kg.). The temperature and pulse have occasionally been slightly raised. *X*-ray films of the skull, spine, pelvis, and chest were normal. The mental condition was that of an anxious woman whose intellectual level is well below average.

Pathological Features

Skin.—Biopsy material from the axilla (Case 1) and from the neck (Case 2) both show similar histological appearances. The main feature is uniform infiltration of the epidermis and corium with monocytoid cells (Fig. 3). The distribution is perivascular in the corium and focal in arrangement in the epidermis. The predominant cells are distinguished by large irregular convoluted nuclei with dense chromatin and scanty feebly acidophilic cytoplasm. Allowing for structural alteration during fixation and staining, these cells are almost identical with those in the blood. The remainder are mainly round cells. The epidermis shows marked parakeratosis, some acanthosis, vesicle formation, and prominent rete pegs. The corium is oedematous and the smaller blood vessels show endarteritis. Sweat glands are present, but hair follicles are conspicuously absent.

Lymph Nodes.—Lymph nodes from the axilla (Case 1) and from the neck (Case 2) are similar in histological appearance (Fig. 4). The capsule is intact, is thickened, and shows no infiltration. The cortex is remarkably broad, slightly lobulated, and paler than normal when stained with haematoxylin and eosin. The cortical follicles are atrophic and compressed, some still showing Flemming's centres. The pallor is due to sheets of cells with large pleomorphic nuclei, often seen in mitosis. Their scanty cytoplasm is faintly acidophilic. Among these cells are small numbers of lymphocytes, eosinophils, and small deposits of melanin. Stained preparations reveal few reticulum fibres in the cortex. Medullary sinus catarrh is marked. The appearances are similar to those of lipomelanic reticulosis ("dermatopathic lymphadenitis"), described by Jarrett and Kellett (1951), although nuclear mitosis and pleomorphism are more marked in the cortical cells of our cases. Jarrett and Kellett thought that the cortical cells of their cases were reticulum cells of histiocyte type. In appearance, though not in depth of staining, their nuclei resemble those of the Sézary cells in the blood of our patients. The variation in staining is probably due to differences in fixation. This view is supported by the presence of large numbers of these cells in lymph-node-puncture smears stained by Jenner-Giemsa technique.

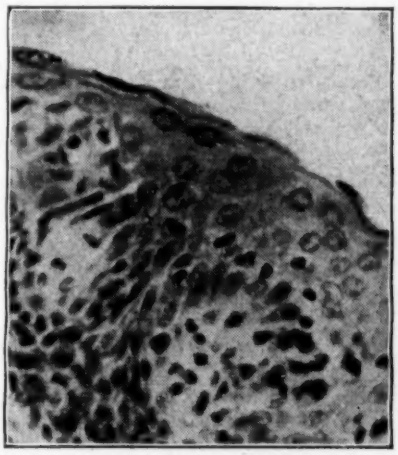


FIG. 3.—Skin section of Case 1 showing monocytoid infiltration. (High-power view.)

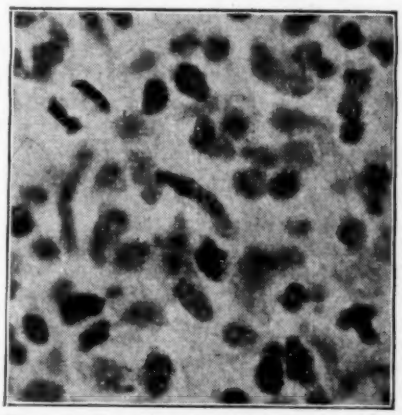


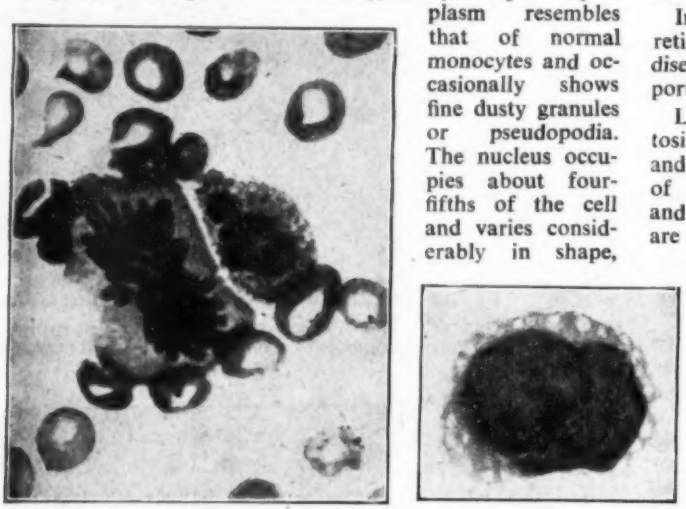
FIG. 4.—Lymph-node histology of the cortex of Case 1 showing Sézary cells in mitosis. (Oil-immersion view.)

Blood Picture.—During observation both patients have shown an unresponsive secondary normochromic anaemia, the Hb varying from 9 to 12 g.% and the M.C.H.C. from 30 to 34%. No nucleated red cells were seen. The platelets appeared normal, ranging from 300,000 to 400,000 per c.mm. At times both patients have shown a slight leucocytosis which never exceeded 16,000 per c.mm., with a varying eosinophilia. The dominant feature, however, was the invariable presence of the *cellules monstreuses* of Sézary. At one time these abnormal cells constituted 35% of the total white-cell count; at others their numbers were so reduced that special search was necessary to detect their presence (Table I).

TABLE I.—*Differential White Cell Counts*

Date	W.B.C.	% Polys	% Lymphs	% Monos	% Eosins	% Basos	% Sézary
			Case 1				
2/6/50	5,300	41	38	3	3	1	14
14/9/50	5,800	54	20	5	3	0	18
30/4/53	7,200	64	18	3	8	0	7
3/6/53	10,000	48	20	3	6	0	23
6/7/53	6,000	66	24	3	5	1	1
24/8/53	14,000	18	43	3	1	0	35
5/1/54	7,500	44	31	7	14	0	4
24/2/54	16,000	47	20	4	9	0	20
			Case 2				
6/1/53	8,000	74	16	6	4	0	<1
20/11/53	10,500	70	10	10	3	1	6
7/12/53	13,000	75	16	2	2	2	3
5/1/54	14,000	75	14	1	3	0	7
24/2/54	12,800	60	13	2	16	0	9

Sézary cells in air-dried Romanovski films (Fig. 5) are approximately twice the size of normal polymorphs. They are peroxidase-negative. The scanty, feebly basophilic cytoplasm resembles that of normal monocytes and occasionally shows fine dusty granules or pseudopodia. The nucleus occupies about four-fifths of the cell and varies considerably in shape, sometimes being round, convoluted, reniform, or bilobed. Binucleate and trinucleate forms have been seen. They simulate mature lymphocytes only in chromatin arrangement and depth of staining. Nucleoli are usually absent or indistinct. These cells were occasionally seen in mitosis in Case 1.



(*a*) (*b*)

FIG. 5.—Peripheral blood: (*a*) Sézary cell in mitosis with adjacent normal polymorphs; (*b*) characteristic mature Sézary cell.

Sternal Marrow.—Marrow smears from both cases show no distinctive features (Table II). Sézary cells are present, but no more than would be expected by dilution with the blood.

TABLE II.—*Myelograms*

Cells	Case 1	Case 2
Blasts	1·0	1·0
Early normoblasts	3·0	4·0
Intermediate normoblasts	10·5	8·0
Late normoblasts	10·5	6·0
Reticulum cells	1·5	0·5
Sézary cells	1·0	3·0
Myeloblasts	0·5	2·0
Promyelocytes	1·5	3·5
Myelocytes	15·5	25·0
Metamyelocytes	20·0	22·0
Granulocytes	25·0	15·0
Lymphocytes	7·0	4·0
Monocytes	1·5	2·0
Plasma cells	0·5	3·0
Megakaryocytes	2·0	1·0

Laboratory Investigations.—The laboratory investigations are summarized in Table III. They have been repeated on several occasions, and the results have invariably been within normal limits, except the E.S.R. (Wintrobe), which is raised slightly in both patients. Hess's test for capillary wall fragility was unsatisfactory because of the state of the skin.

TABLE III.—*Laboratory Investigations*

Investigation	Case 1	Case 2
W.R. and Kahn test	Negative	Negative
Serum albumin	4·1 g.%	2·8 g.%
Serum globulin	2·4 g.%	2·2 g.%
Total protein	6·5 g.%	5·0 g.%
A/G ratio	0·6/1·0	1·3/1·0
Colloidal gold	0	0
Alkaline phosphatase	7 (K.A. units)	20 (K.A. units)
Serum bilirubin	0·5 mg./100 ml.	0.5 mg./100 ml.
Plasma prothrombin time	12 secs. (control 15)	15 secs. (control 15)
Whole blood-clotting time	5 mins. (L.W.)	5 mins. (L.W.)
Fasting blood sugar	62 mg./100 ml.	65 mg./100 ml.
Blood cholesterol	250 " "	150 " "
Blood urea	25 " "	25 " "
Serum Na, K, Cl	320, 20, 620 mg./100 ml.	330, 19, 610 mg.
E.S.R. (Wintrobe)	31 mm./hr.	20 mm./hr.
24-hour 17-ketosteroid excretion	4·7 mg. (920 ml.)	7·9 mg. (725 ml.)
24-hour creatinine excretion	880 mg.	850 mg.
Urine	N.A.D.	N.A.D.
B.M.R.	+1	+25

Diagnosis

The features of Sézary's syndrome are intense pruritus, generalized erythrodermia, pigmentation, and superficial lymphadenopathy, associated with the presence of unusual monocytoid cells in the blood.

In general this description might apply to many of the reticuloses, of which "lipomelanic reticulosis," Hodgkin's disease, the leukaemias, and mycosis fungoides are of importance.

Lipomelanic reticulosis often starts as a localized dermatosis, before generalized erythrodermia, intense pruritus, and superficial lymphadenopathy develop. Varying degrees of exfoliation, lichenification, pigmentation, excoriation, and low-grade sepsis occur. Attacks of profuse sweating are almost invariable. Eosinophilia is usually present. It may be difficult to differentiate this condition from Sézary's syndrome without careful haematological examination.

Hodgkin's disease frequently presents with lymphadenomatous masses, and less often with cutaneous manifestations. When it affects the skin pruritus may be marked, but erythrodermia is unusual. Prurigo papules or scratch marks may be the only visible signs.

The leukaemias should always be suspected. The cutaneous manifestations may take the form of intense pruritus, erythrodermia and exfoliation, although nodules and areas of infiltration occur. The hair and nails may shed. Purpura often occurs in the acute phase, the patient usually having an irregular pyrexia. Erythrodermia in the form of glazed or weeping areas may give the classical picture of "l'homme rouge." Both lymphadenopathy and hepato-splenomegaly may be present. The characteristic blood and marrow pictures indicate the diagnosis.

Mycosis fungoides is usually substantiated only when tumour formation has occurred. The earlier lesions are often red scaly patches, transient at first, eventually becoming fixed and infiltrated. Involution may occur, leaving pigmented areas, but persistence is usual. They usually precede the development of cutaneous tumours by a long interval.

Parapsoriasis embraces an ill-defined group of conditions, of which parakeratosis variegata may possibly cause confusion. Although generalized retiform hyperaemia and scaling are present, subjective sensations and lymphadenopathy are absent, and the blood picture is normal.

In cases of generalized erythrodermia with lymphadenopathy, chest *x*-ray and haematological examination, including marrow puncture, and biopsy of the skin and lymph nodes should be done. If doubtful, these examinations should be repeated at intervals.

Discussion

We have known of this syndrome now for two years. Despite the study of likely patients, only two cases have been seen, and the literature contains only five other confirmed cases. The condition therefore appears to be of some rarity. This is perhaps due to failure to recognize

TABLE IV.—*Clinical Features of Published Cases*

Feature	Sézary and Bouvrain (1938) Case 1	Sézary Horowitz, and Maschas (1938) Case 2	Baccaredda (1939) Case 3	Sézary and Bolgert (1942) Case 4	Wilson and Fielding (1953) Case 5	Present Case 1 (1954) Case 6	Present Case 2 (1954) Case 7
Age	58	69	67	66	67	77	40
Sex	F	F	F	F	M	F	F
Duration	18 months	30 months	36 months	39 months	? 16 years	8 years	6 years
First symptom	Pruritus	Pruritus	Pruritus. ? fissures soles and palms	Pruritus	Transient rashes. Pruritus	Pruritus	Pruritus
Erythrodermia and scaling	+	+	+	+	+	+	+
Pigmentation	+	+	+	+	+	+	+
"Mottling"	+	+	–	+	+	+	+
Purpura	+	–	–	+	+	+	+
Eruption present	Papular	Papular	?	?	–	–	Eczematous
Cutaneous infiltration	+	+	+	+	+	+	+
" oedema	+	+	+	?	+	+	+
Pyodermia	+	–	+	?	–	+	+
Sweating attacks	+	–	–	–	–	–	–
Hair	Alopecia 19 years	Alopecia	Thicker and blacker	Alopecia	Normal (sparse)	Alopecia	Alopecia
Mucous membranes	Ectropion	Normal	Ectropion. Pigmented	Normal	Normal	Ectropion	Normal
Superficial lymphadenopathy	+	+	+	+	+	+	+
Hepatosplenomegaly	?	?	Terminal liver enlargement	–	–	Liver just palpable	Liver palpable
X-ray chest	Normal	Normal	Normal	Normal	Normal	Normal	Normal
General condition	Poor	Poor	Moderate—death unexpected	Poor	Poor	Good	Moderate

the syndrome. The case reported by MacCormac and Whitby (1936) as leukaemic erythrodermia was possibly an example of this condition. Hodgkin's disease with cutaneous manifestations and circulating Reed–Sternberg cells (Symmers, 1924; Miller, 1928; Wigley, 1937; Combes and Bluefarb, 1941) has perhaps been confused with Sézary's syndrome because of the superficial similarity of Reed–Sternberg cells to the *cellules monstreuses*.

Review of the clinical features of the five published cases and the two reported here shows that this condition seems to occur mainly in elderly females (Table IV). Some patients given a history of skin trouble, such as transient rashes, occurring some time before the onset of pruritus, which was the presenting symptom in all cases. Pruritus was intense, eventually generalized, and later associated with exfoliative erythrodermia, pigmentation, purpura, and "mottling," which in one patient (Case 1) took the form of a retiform telangiectasia.

Cutaneous thickening and oedema give a strange facies, which may be accentuated by ectropion, patchy loss of hair, or even total alopecia. Other features noted include dystrophy of the nails, arthropathy, frictional eczema, excoriation, and pyodermia. In all cases superficial lymphadenopathy was marked. Chest *x*-ray examination revealed no evidence of mediastinal glandular enlargement.

The general condition of one of our patients (Case 1) has consistently remained good, although the local condition has varied; the other patient has been in poor health for some time. At intervals, both patients have had short courses of A.C.T.H. and cortisone with temporary improvement, but relapse has invariably occurred. At present both are improving spontaneously, and further A.C.T.H. is therefore being withheld. Pigmentation, erythrodermia, and scaling are becoming less marked, and there is associated diminution of the superficial lymph nodes. The eyebrows and eyelashes are regrowing. The mental outlook of both patients has improved.

The number of cases reported is inadequate to assess the prognosis. It is possibly significant that all except one case have been elderly. Sézary does not state the actual cause of death in his cases. In Baccaredda's case deterioration and death were unexpected, and due to pyelonephritis; Wilson and Fielding's patient died from cardiac disease. At present the general condition of our cases appears satisfactory and early death is not anticipated. Apparent clinical improvement in our cases whilst in hospital may not indicate that the condition is any the less malignant, as spontaneous remissions occur in all reticuloses.

The clinical and laboratory features of the present cases and of those reviewed by Sézary (1949) indicate that they form part of the reticuloses. Although Sézary described the condition as a "nouvelle réticulose cutanée," the cytological appearances are similar to those of the reticulum-cell reticuloses (Israëls, 1951) although affecting different structures.

One of us (R. L. T.) has had the opportunity of examining blood and marrow smears from the cases described by Israëls (1951) and classified by him as reticulum-cell reticulosis. In some of these cases the abnormal cells closely resemble the *cellules monstreuses* of Sézary. The difference between these series appears largely clinical, possibly depending on the anatomical site where the disease begins.

In general, the present classification of the reticuloses is unsatisfactory. It comprises a large group of conditions, varying from the lipoid storage diseases to disturbances characterized by progressive proliferation of all or part of the reticulo-endothelial tissues. In the British literature two classifications, depending respectively on the histology (Robb-Smith, 1938) and the clinico-pathological features (Israëls, 1953) are recognized. Israëls suggests that in his classification many of the subgroups delineated by histological examination alone may be but phases in the ultimate evolution of the same disease process.

With this in view, and because of the similarity of the lymph-node histology of our cases to that of lipomelanic reticulosis, we have reviewed the series reported by Jarrett and Kellett (1951) from this hospital in order to see if those surviving showed Sézary cells in the blood, or other evidence of malignant reticulosis. Of their 16 patients (13 men and 3 women), who were observed for 3½ years, 5 have since died, and one could not be traced. Of the remainder, one is still affected and 9 are clinically clear. Sézary cells were not found in any of these cases, nor were other manifestations of reticulosis. In no case was the certified cause of death related to the reticuloses.

Sézary provisionally classified the *cellules monstreuses* as aberrant reticulum cells originating from the reticulum tissue in the skin. This view is probably correct, although we are less certain of the site of origin, as they resemble the proliferating reticulum cells of lymph nodes—smears from which in the present cases show a high proportion of these cells. Their similarity to the proliferating cells seen in the blood and marrow of Israëls's reticulum-cell reticulosis is again emphasized.

Histologically, this appears to place Sézary's syndrome as a transitional phase between lipomelanic reticulosis and reticulum-cell reticulosis.

The apparent benign clinical course of lipomelanic reticulosis (Pautrier and Woringer, 1937 ; Jarrett and Kellett, 1951) contrasts sharply with the typical fulminating course of reticulum-cell reticulosis. The review of Jarrett and Kellett's lipomelanic series indicates that nine patients have recovered —so much so that this diagnosis would not now be entertained.

Clinically, therefore, the present cases appear to lie between these extremes, having run a chronic course for several years. Although at present showing some spontaneous remission, it is possibly significant that Case 1 has slowly deteriorated since first seen.

Wilson and Fielding (1953) were hesitant about applying the term "reticulosis" to Sézary's syndrome. They suggest that it is similar to lipomelanic reticulosis in that it represents a non-specific response to cutaneous disturbance which "is not, at least to begin with, malignant in character." We consider that Sézary's syndrome is more than simple reactive hyperplasia. It is an early phase of malignant reticulosis primarily affecting the skin and superficial lymph nodes. Involvement of the deeper structures is probable provided death does not occur from intercurrent disease.

The ultimate aetiology of this syndrome, as of all reticuloses, remains obscure. Careful study of prolonged conditions such as this, linking reactive states to irreversible malignancy, may possibly contribute to knowledge of the latter.

Summary

Two further cases of Sézary's syndrome are described and the five previous cases reviewed.

It is thought to be a local reticulosis intermediate in phase between benign reaction and frank malignancy.

The chronicity is possibly due partly to its "pre-malignant" character and partly to the organ affected—the skin and superficial lymph nodes.

We are grateful to Dr. S. Varadi, of Sheffield, and Dr. M. C. G. Israëls, of Manchester, for advice and encouragement; to Mr. L. Sutcliffe, of Bradford, and Mr. C. Howe, of Manchester, for the photographs; and to Miss M. E. Whyte for technical assistance.

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Speaking at a graduation ceremony for medical students at St. Andrews University on January 14, Principal T. M. KNOX, vice-chancellor of the University, uttered a warning against the modern tendency to divert professional men from the practice of their profession into administration. "It used to be said in scorn about teachers," he said, "that they taught only because they could not do. One might perhaps have expected an equally scornful remark about administrators." But administration now presented itself in such an attractive guise that many professional men were lured away from the exercise of their professional skill into an office chair. "I am not sure that this is a sign of health in the body politic." "In fact," Principal Knox went on, "administration is just a necessary evil, not an end in itself, but a means to the freedom and progress of professional work, and, whatever the difficulties of administration may be, it is a great deal easier than the exercise of professional skill."—*Scotsman.*

INCIDENCE OF DENTAL CARIES IN CHILDREN UNDER 5 YEARS OLD

BY

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Amongst the few surveys of the incidence of dental caries in children under 5 years old that have been published in this country are those of Knowles (1948) and Jackson (1952). Frequent reports have been made on the dental condition of children aged 5 and over, in particular the studies of Mellanby and Coumoulos (1944, 1946) and Mellanby and Mellanby (1948, 1950, and 1951).

In 1948 it was decided to carry out a series of surveys of the dental condition of the children in the city of Liverpool day nurseries. Four static surveys were completed during the period 1948–52, and the findings are presented in this paper.

The primary aim of these surveys was to determine the incidence of dental caries in children under 5 years old, before they passed into the regular care of the school dental service. A high caries attack rate would, without doubt, prejudice the chances of maintaining the dental fitness of these children by adding to the already heavy burden of dental work now being undertaken. Further, it would be possible to estimate on the basis of the results the number of dentists required to maintain a satisfactory treatment service.

As the second aim, it was desired to ascertain whether any variation in incidence of dental caries would occur over a period of years and to speculate upon possible causes of such variations. For example, a poor state of dental health might be attributed to the possibility of dietary errors during pregnancy and lactation affecting the resistance of tooth structure to caries attack. Alternatively, or in addition, post-natal dietary errors might create in the child environmental conditions in the mouth which would favour the carious process.

Children under 5 provide a useful measure of pre- and post-natal influences. The deciduous teeth begin to be mineralized *in utero* during the eighth week. The completion of the mineralization of the deciduous dentition occurs after birth, during the third year. At about 6 years of age the first permanent molar teeth erupt in a position distal to the deciduous second molar. This is followed shortly afterwards by the shedding of the deciduous incisors. Thus, by confining the investigation to children under 5 the complications of mixed (deciduous and permanent) dentitions are avoided.

Materials and Method

Liverpool Day Nurseries.—These are administered by the maternity and child welfare department under the city medical officer of health. In 1950 the number of day nurseries was increased from 13 to 15, and in 1953 it was reduced to 14. The nurseries are located in widely separated areas, which vary from the poorer districts to the more salubrious parts of the city. The number of children accommodated on a daily basis varies from 40 to 70. The eligibility of children for places in the day nurseries depends on the circumstances of the applying parent. The object is to provide daytime nursery facilities which will enable the parent to go out to work. At one time children who had been accepted could remain at the day nursery until school age, but this policy

was changed in 1951. It is now the intention that accommodation should be available for restricted periods of time until the parents can make other arrangements. The estimated annual turnover is about 15%. The nurseries open at 6.30 a.m. for the admission of children and close at approximately 6.30 p.m., after the children have been collected by the parents. At present the daily charge is three shillings.

Feeding Arrangements.—The children are given three meals at the nursery. Breakfast and tea are prepared and cooked at the individual nurseries. However, the midday meal is prepared, cooked, and then delivered in insulated containers from central kitchens by the school meal service. The vegetables are cooked at the nurseries. In addition, the children receive cod-liver oil, orange juice, and one pint (570 ml.) of milk daily. There are no between-meal snacks whilst the children are at the day nursery. The only exception is that sweets of the dolly mixture type are given after inoculations. Incidentally, many of the children arrive eating sweets, buns, or cakes, and often are given some form of confection by the parents on leaving the nursery at the end of the day. Knowles (1948) reports a similar practice in the London area nurseries. The reason for this may be the desire of the parents to establish or re-establish themselves in the affections of these very young children after long daily separations. It is known, too, that many of the children are similarly indulged at the week-ends.

Dental Standards.—All examinations were carried out by me, using mirror and probe in a good light. New Ash's Clinic No. 17 probes were used, being discarded after 15 to 20 examinations. The probe is fairly coarse, with a short taper to its point. I recorded findings on a simple chart. Caries was recorded, showing the surface(s) of the tooth involved, and graded as follows: Grade 1: cavities which would definitely admit the point of the probe; doubtful cavities were not included. Grade 2: cavities involving up to one-third of the crown of the tooth. Grade 3: cavities involving more than one-third of the crown; missing teeth due to extraction were placed in this category. Fillings were recorded as caries and scored accordingly. The condition of the gums was also recorded, and the severity of gingivitis was graded 1, 2, and 3. Co-operation was enhanced if the examinations were carried out in the presence of as many children as possible. The older children were usually examined first; the younger and doubting children usually followed without demur. Only four toddlers resisted all manner of coaxing to allow examination during the five-year period. Much of this success was due to the kind yet firm handling of the children by the matrons and their excellent staffs. A simple method was used to examine the babies. I sat facing the nurse and the baby was placed on our knees with its head towards me. This inversion of the baby proved an easy method of obtaining a view of the upper teeth.

Numbers Examined.—The number of children examined is given in Table I. In the first two examinations only those children were examined who were likely to stay a reasonable length of time. This accounts for the low figures in the 4–5 age groups in the 1948 and 1949 examinations. When the longer stay policy was abandoned a progressive survey was no longer possible. Thereafter all children were examined.

TABLE I.—*Children Examined*

Age	Dec., 1948	May, 1949	June, 1951	May, 1952	Total
Under 2 ..	33	21	151	152	357
„ 3 ..	146	136	153	197	632
„ 4 ..	117	149	134	148	548
„ 5 ..	28	52	156	136	372
Total ..	324	358	594	633	1,909

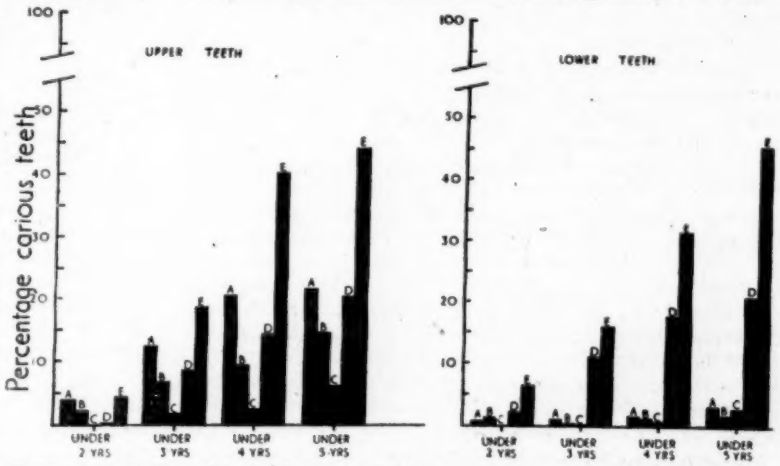
Results

The results of the series of four examinations are presented by age groups (Table II). The percentage of caries-free children in the series of examinations shows a similar trend for all age groups. There was a definite drop in the percentage in May, 1949, followed by a rise in June, 1951, to a higher level than in December, 1948.

The average D.E.F. (decayed, extracted, and filled) teeth per child has been calculated. The trend of caries attack is similar.

The effect of a small number of children each having a large number of decayed teeth would be to raise the overall average of D.E.F. and perhaps show a false position. This effect can be minimized by expressing the findings in terms of D.E.F. per 100 teeth. However, the trend is again similar.

The susceptibility to decay of individual teeth varies. The results of all four examinations have been pooled, and are presented by age groups (see Chart). There is a steady rise, though at different rates, in the susceptibility of all teeth with the increase in the age of the children. The pattern of the results approximates the findings of Knowles (1948). The high susceptibility of the deciduous molars, especially the second



Caries susceptibility of individual teeth by age groups for both upper and lower arches. A=central incisor. B=lateral incisor. C=canine. D=first molar. E=second molar.

TABLE II.—*Results of Four Surveys*

Date Examined:	Dec., 1948				May, 1949				June, 1951				May, 1952			
Age:	–2	–3	–4	–5	–2	–3	–4	–5	–2	–3	–4	–5	–2	–3	–4	–5
Percentage caries-free children ..	84·8	58·9	29·0	14·8	76·2	52·5	25·8	5·9	91·5	70·2	40·1	24·0	90·6	66·8	39·0	27·9
Average D.E.F. per child ..	0·3	1·1	2·2	5·0	0·6	1·6	2·6	4·7	0·2	1·3	2·4	4·0	0·2	1·8	3·5	4·1
D.E.F. per 100 teeth	1·8	5·8	10·8	23·7	3·4	7·9	14·3	22·2	0·9	7·7	12·4	20·6	1·6	7·18	18·9	20·4

D.E.F.=Decayed, extracted, or filled teeth.

molars, is particularly striking when considered in terms of the length of time these teeth are exposed to the oral environment. It is generally accepted that the eruption times of the deciduous dentition are: incisors, 6–10 months; canines, 16–20 months; first molars, 12–16 months; and second molars, 20–30 months (Kronfeld, 1939). Occasionally eruption times fall outside these dates. It is possible that enamel formed at about the time of birth is less resistant to caries. Rushton (1933) has shown that the disturbance of birth causes a change in the enamel. Histological sections show a line of demarcation between the pre-natal and post-natal enamel. It is an appalling thought that 7% of the second deciduous molars are decayed before the children are 2 years old, and that, even worse, over 45% are decayed before the children reach 5 years. Comment is made below on this point.

The severity of caries was assessed in accordance with the grades referred to above. The results of the four examinations for all age groups have been pooled. Of the 876 decayed first molars, 28% were grade 1, 46% grade 2, and 26% grade 3. Of the 1,808 decayed second molars, 43.5% were grade 1, 38.5% grade 2, and 18% grade 3.

Finally, only 11 children out of the total number of 1,909 had any fillings present. The gum condition of the children was generally good, and less than 2% suffered from clinically detectable disturbances.

Discussion

The primary object of the survey, which was to ascertain the incidence of caries, has been achieved. It is reasonable to comment on these findings relative to all children, for it has not been shown that dental caries is any less prevalent at either end of the social or financial scale. The only exception is that children fully resident the year round in orphanages, institutions, and the like have a much lower incidence of decay (Hewat, 1931, 1932). These latter children constitute only a very small fraction of the child population of the United Kingdom.

It is a sad reflection that all children who enter school have, on an average, five decayed teeth each. Thus the school dental service is faced with a considerable problem.

The live-birth rate in Liverpool over the past ten years averages approximately 16,500 a year (A. B. Semple, 1953, personal communication). This provides a basis for the calculation of the number of dentists required. Thus, 16,500 × 5 decayed teeth = 82,500 teeth requiring treatment in the 4–5-year-old children alone. It is possible for one full-time school dentist to complete, say, 3,168 fillings a year. Assuming one simple filling per tooth, 26 dentists would be required to deal only with this 4–5-year-old age group each year. Certain factors would affect this figure, such as extractions, root fillings, multiple fillings, difficult children, refusals, attendances to private dentists, etc. Even if allowance be made for these factors, the situation is impossible. In Liverpool the establishment of dental surgeons in the school service is 24, to deal with the whole school population and such pre-school children who do attend.

No doubt much the same situation exists in the majority of other similar communities. As a result multiple extractions of the deciduous teeth have to be carried out so that time may be devoted to the preservation of the permanent teeth of the older schoolchildren. The consequence is that large numbers of children become dental cripples with impaired mastication and malocclusion of the permanent teeth due to the early loss of the deciduous teeth. The deciduous molars should be retained until 9–10 years old, when the permanent premolar teeth which follow them are due to erupt. It is little wonder that the demand for the treatment for these irregularities far outstrips the supply of trained orthodontists. Nor is it surprising that full dentures are required by adults at an early age.

The increase of dental man-power, though essential to a certain level, does not provide a satisfactory solution to this problem. The real solution lies in more dental research into the aetiology of dental caries, and, in the meantime, the application of every known measure which may bring about a reduction in decay.

The second object of these surveys was the examination of the trend of caries attack during the five years. The result is interesting (see Table II).

It is reasonable to assume that the influential factor responsible for this trend was not of pre-natal origin affecting tooth structure during formation. A common pre-natal factor would not have caused similar variations in all the age groups at the same time. Hence it is reasonable to examine the possible local or environmental factors which might be concerned.

Diet.—There were no significant changes in the main nutritional parts of the diet during the period. Some improvement in quality was introduced during 1951. No correlation is possible.

Supplements.—The prescribed amounts and quality of the vitamin supplements were maintained. At the nurseries the daily pint of milk was given regularly. Changes in the supplying contractors precluded any basis for correlation.

Sugar and Sweets.—The variations in the ration between 1942 and 1953 were examined. This is discussed below in detail.

Oral Hygiene.—No changes in the oral hygiene routine at the nurseries were made during the period.

Extraction Rate of Flour.—No correlation.

Availability of Fruit and Vegetables.—Usual seasonal variation; comment is made below on this point.

The presence of easily fermentable carbohydrate in the mouth has for some time been thought to be an important factor, and must be considered. Stones *et al.* (1954) completed a two-year study in 1951 on the effect of administering 22 oz. (620 g.) of sugar in addition to the ration. The extra sugar was given at meal-times only. There was no significant difference in the incidence of dental caries between the children receiving the extra sugar and those in the control group. On the other hand, the recent comprehensive studies carried out at the Lund Research Station at Vipeholm Hospital in Sweden are important, for the between-meal carbohydrate has been shown to be one of the most important factors in causing dental caries (Gustafsson *et al.*, 1952).

It is tempting, therefore, to speculate regarding the effect of the United Kingdom rationing of sugar and sweets during the period of the four surveys.

The May, 1949, examination, which revealed a drop in the percentage of caries-free children, took place about two to three weeks after the derationing of sweets on April 24, 1949. It may be remembered that there was such a wave of buying that plenty soon became a famine after a few weeks, and rationing was reimposed on August 14, 1949. Thereafter the size of the ration varied until derationing on February 5, 1953. It is interesting to consider whether the quantities of sweets obtained by these children during this period were sufficient to produce changes in the oral environment which hastened the carious process.

Little is known about the rate of development of carious lesions. It is a common clinical experience to find fresh carious lesions within a three-months period after children have been made dentally fit. In this case two weeks seems a short time for the development of lesions; nevertheless, speculation is provoked. Dental surveys of large numbers of children need to be undertaken every two to three weeks over a period of at least two years. Two problems may then be solved—the rate of development of clinically detectable caries, and the possible seasonal variations in the caries attack rate.

Careful study of the former must necessarily precede that of the latter. In the United States of America the greatest incidence of caries appears to occur in the autumn and winter, and the lowest incidence in the summer and spring (Price, 1930; McBeath and Zucker, 1938; Lathrop, 1941, 1943). This finding is not in agreement with the present study, and may be due to local differences between this country and America.

The seasonal variation, if it occurs, could be due to the variations in the composition of diet such as increased consumption of sweets during the periods when there is less inclination to eat fruit and vegetables or when supplies of the latter are restricted by supply or price. If the cause of such variation could be found, a real advance would have been made in dental research.

Conclusions

Children under 5 years suffer severely from dental caries and are in need of dental care. With any foreseeable available dental man-power in the next few years, the task is too great to be dealt with on a reparative basis, so that every possible preventive measure needs to be applied.

At the present date two measures are of proved value in reducing the caries attack rate. The fluoridation of domestic water supplies in the United States is so effective that the United Kingdom Mission of the Ministry of Health (1953) has recommended the inauguration of studies in this country.

The second measure, the plain water rinse after all food and drink, is of individual application. Thus to make this measure effective the education of all those having children in their care is essential. The medical profession is in a unique position in this respect. For it is to the medical practitioner that parents, teachers, and heads of institutions turn for guidance concerning the care of very young children.

The habit of drinking plain water after eating, whatever it may be, must be established in children before the first teeth erupt. As soon as possible they must be taught to rinse and to swallow the water. Experience has shown that children learn this procedure more quickly than when expectorating the water. In any case, the former method is the more socially acceptable. Three rinses should be carried out, forcing the water between the teeth to dislodge the food debris. This routine must never be varied. It must continue throughout the school terms and holidays alike, immediately after all meals, mid-morning or afternoon snacks, sweets, chocolates, and so on. Fibrous foods, such as apples, at the end of meals enhance the procedure. When possible the rinsing should be followed by tooth-brushing, but it must always be done after the last food or drink is taken at night.

I have prepared an instructional pamphlet (United Liverpool Hospitals, 1953). It is written in the simplest terms with deliberate repetition, and is designed to introduce this procedure of preventive dentistry to the mothers of very young children. Actually, the instructions apply to children of all age groups. The method provides, short of a return to primitive diets (Price, 1939), a logical means of reducing the caries attack rate, by the removal of the carbohydrate substance which is so essential to the existence of the organisms associated with the caries process. The regime is simple and clear enough for all to understand.

Summary

The findings of four surveys, during the period 1948–52, of the incidence of dental caries in children under 5 years old are presented.

These children suffer considerably from dental caries. Children on reaching school age have an average of five decayed teeth.

The school dental service faces an impossible task in maintaining a reparative service with the present numbers.

The results show that the trend of dental caries incidence follows a similar pattern in all age groups under 5 years. There was a drop in the percentage of caries-free children in 1949 compared with 1948, 1951, and 1952. An attempt is made to speculate on the cause of this drop.

In the conclusions that are drawn two preventive measures of proved value are mentioned. The fluoridation of domestic water supplies in the United States has been of such value that the United Kingdom Mission has recommended the initiation of fluoridation studies in this country. The second measure recommended is the plain water rinsing of the mouth after eating or drinking anything whatsoever. It should be taught to children before teeth erupt. The technique is described.

The education in dental health of all those who have children under their care is a primary need, whether they be the parents, teachers, or heads of institutions. The medical practitioner is in a unique position, for it is from him that guidance is sought initially in the dental care of these young children.

I wish to express my thanks and gratitude to Professor W. M. Frazer, late medical officer of health for the City of Liverpool, and Dr. Ruby M. Bell, late assistant medical officer of health in charge of the maternity and child welfare department, who kindly gave me facilities to carry out these surveys. Also to their successors, Professor A. B. Semple and Dr. V. J. M. Stark for their continuing interest. I wish to thank the supervising matron and all the matrons and staffs of the individual day nurseries who have helped so much in carrying out the work, Mr. W. Lee for photographing the chart, and Miss I. Rayner, who carried out the tedious task of collating the data for analysis. I am indebted to the Ministry of Food, Liverpool, who kindly supplied the sugar and sweet ration figures from which some data were obtained. Finally, I express my appreciation to Professor H. H. Stones, Director of Dental Education, School of Dental Surgery, University of Liverpool, for his help and encouragement.

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The PRINCESS ROYAL unveiled a plaque at No. 10, South Street, Park Lane, London, on January 21 to commemorate the residence in a house on that site of Miss Florence Nightingale. She lived there from 1865 until her death at the age of 90 in 1910. The proposal to erect the plaque was made to the London County Council's planning committee by the Florence Nightingale Hospital, the National Council of Nurses, and the Western Heritable Investment Co., Ltd., the occupiers of the present building. The Princess Royal, who is Patroness of the Florence Nightingale Hospital, was received by Sir CULLUM WELCH, chairman of the hospital, and by Lady WELCH and Mrs. A. C. E. KIMBER, joint chairmen of the hospital's appeal committee. A link with Miss Nightingale herself was provided by the presence at the ceremony of Mrs. ALICE CRAWLEY, who as Miss Cochrane was her secretary for some years. Also present were the chairman of the board of governors, St. Thomas's Hospital, and members of the executive and appeals committees of the Florence Nightingale Hospital, representatives of the National Council of Nurses of Great Britain and Northern Ireland, and the chairman of the London County Council.

A CASE OF BILATERAL FATIGUE FRACTURE OF THE FIBULA

BY

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Fracture of a bone may result from ordinary functional activity in the absence of any obvious injury or violence. Such fractures are referred to as fatigue or stress fractures, and occur spontaneously in apparently normal bone as a consequence of the summation of stresses which singly are insufficient to produce fracture (Burrows, 1948). The incidence of this condition rose sharply during the second world war, but since then, as judged by published cases, it would appear to be an infrequent finding. By far the commonest site of this form of fracture is in one of the metatarsal bones; the tibia is involved five or six times less frequently, and the fibula forty times less frequently (Hartley, 1943). The series of 32 fatigue fractures analysed by Richmond, 1945, showed the following locations: metatarsals, 25; upper third of tibia, 5, and lower third of fibula, 2.

The case reported below is an example of fatigue fractures involving the lower third of both fibulae in a middle-aged housewife.

Case History

A married woman aged 45 was referred to one of us in a general medical clinic as a case of "arthritis of ankles." She complained of pain and swelling in both ankles. The pain had been noticed before the onset of the swelling, and had gradually become very severe—so much so that walking had become difficult. She lives in an upstairs flat and runs up and downstairs to and from a shop many times a day. The only unusual exercise undertaken recently has been that involved in using a treadle sewing-machine employing her right foot only. In addition she runs the flat for herself and her husband.

On examination there was a visible swelling 1½ in. (3.8 cm.) above the tip of the lateral malleolus on the right side. There was a tender bony thickening at this level on both sides. Pain could be elicited at these sites by everting or rotating the calcaneus in the ankle mortise. Similar pain was produced by fully dorsiflexing the foot at the ankle-joint. Both heels showed eversion when standing. Radiographs showed fractures of both fibulae 1½ in. (3.8 cm.) above the malleolar tips, slightly oblique in direction as seen in the antero-posterior views. The lateral views showed little of the fractures. Callus was visible on both sides (see Figs.). No osteoporosis was evident in these films or in films of the hands.

Stirrup strapping was applied to both ankles and the patient was advised to curtail her activities as much as possible. Her signs and symptoms gradually improved, and four months later her pain had almost disappeared, and only slight bony thickening was apparent clinically.

Comment

The diagnosis of fatigue fracture rests on a consideration of the clinical features and radiological findings. Pain is the usual presenting symptom, and may be of acute onset or of gradually increasing severity. There is little in the patient's history to indicate the nature of the condition; since no record of injury or violence or of a sensation of snap is forthcoming, the possibility of fracture may well not arise.

The local signs are those of localized swelling and tenderness. Burrows (1948) has described three cases of fatigue fracture of the fibula in which tenderness was absent and five in which swelling was not evident. Oedema over the area of the dorsum of the foot may be present and local redness and increased temperature are other occasional findings. In unilateral examples limp does not appear to be a significant sign, but it was certainly a prominent feature in our case. Movement of the ankle-joint is unimpaired, but passive eversion of this articulation produces severe pain over the site of the fracture. Similar pain is produced by any movement which causes widening of the ankle mortise, such as full dorsiflexion of the foot.

Radiograph showing fracture and callus formation of both fibulae.

Watson-Jones (1952) emphasizes that radiographs may show no abnormalities until the fissure is widened by reactionary osteoporosis, or until bone is laid down beneath the periosteum. Burrows states that a fracture line may be evident in some instances only at the end of the third week, and that even then it may be recognized only with the aid of a lens; a dense band is often the earliest change in the *x*-ray picture, and may be the last abnormality to clear. At a later date callus appears, and may consolidate and persist for many months as a bony buttress across the concavity or "stressed" aspect of the bone.

From his review of the literature and his own personal series of cases Burrows was able to segregate two groups in which fatigue fractures of the lowest third of the fibula occur. The first group comprises young athletes, such as runners and skaters. It is not difficult to understand the mechanism of production of the fracture or fractures in such activities. The second group, to which our own case belongs, is made up of women of middle age or over who are much on their feet in the course of the day. The aetiological factors in this second group are probably several. Burrows refutes the suggestion of Hartley (1943) that osteoporosis plays any significant part in its causation.

The site of the fractures would seem to offer an explanation of the mode of production; in our case they were situated 1½ in. (3.8 cm.) above the tip of the lateral malleolus, and a similar level was noted by Burrows. Here the bone is largely cancellous in type and the fracture line is just below the interosseus ligament. Continued eversion of the feet, particularly in overweight females, will throw a strain on the lateral aspect of the fibula. The pull of the interosseous ligament medially and the forcing of the lower portion of the fibula in an outward direction will determine a focus of stress concentration at a point immediately below the ligament. It may well be, therefore, that at this focus deformation occurs which is at first elastic, and so reversible, but which later becomes permanent. As a consequence of

the localized molecular disruption there results a gradual spread towards the centre. The analogy to fatigue fractures in metals presents itself and would repay further study. It would be interesting to note whether the characters of the fractures in both types are similar. In those of metals the fracture is comparatively smooth at its outer zones where the surfaces have been rubbed together on innumerable occasions prior to the sudden spread of the fracture transversely, this suddenly fractured zone showing relative coarseness. Difficulty remains in affording an explanation for the predominance of this group of fatigue fractures of the fibula in females.

Summary

A case of bilateral fatigue fractures of the fibula in a middle-aged woman is reported.

Point tenderness, and pain at the same site when the ankle mortise was widened by everting the heels or dorsiflexing the foot, were features of the clinical findings.

The site of the fractures was identical with that described by Burrows (1948) in women of middle age.

Treatment advised was stirrup strapping and rest.

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INTUSSUSCEPTION OF VERMIFORM APPENDIX IN ALLERGIC CHILDREN

BY

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Chronic intussusception is relatively rare in childhood and the symptoms are often ill defined, especially when the appendix is the site of intussusception. The following two cases are of interest because the presence of allergic disease caused difficulty in diagnosis. The age of the second patient makes her case additionally interesting, because this is only the second report of intussusception of the appendix in a child under 2½ years of age, the first being that of a boy aged 10 months (Forshall, 1953).

Case 1

A boy of 3 years was sent to the Royal Hospital for Sick Children because of attacks of diarrhoea with blood in the stools. A tentative diagnosis of gastro-intestinal allergy had been made because there was a history of infantile eczema.

After spontaneous delivery at term the child was breast-fed for three weeks and then put on to cow's milk, following which he developed eczema. He had bronchitis at 6 months and measles at 1 year. There was no family history of allergic diseases. The patient was well until seventeen days before admission, when he passed a small quantity of bright red blood per rectum. Two days later he had a little diarrhoea and again passed blood. Treatment with a chalk mixture was prescribed and no further bleeding occurred for ten days. Five days before admission he passed several loose stools containing a larger amount of blood; he looked pale but not ill. On the day of admission he had an attack of abdominal pain and passed more blood in the stool.

Examination on admission showed a poorly nourished child with rough dry skin. A soft systolic murmur was heard at the apex of the heart, otherwise physical examination was negative. Blood examination revealed a moderate hypochromic anaemia.

On the day following admission he passed a dark stool, and a small, smooth, mobile mass was felt in the right iliac fossa which disappeared and reappeared at intervals during the next two days. He passed no further stools, but complained on one occasion of abdominal pain lasting for a few minutes. On the fourth day a tumour was felt extending from the right iliac fossa to the right hypochondrium, and at operation (Miss R. M. Mackay) a mass was found consisting of the caecum, into which the appendix had completely intussuscepted. There was no obvious impairment of the blood supply, but all layers were so oedematous that reduction was impossible. The caecum was opened, and the completely inverted and very swollen appendix was seen inside; the tip was just becoming gangrenous, but the rest was only congested. The appendix was removed and the caecum closed. Following blood transfusion, recovery was uneventful.

Case 2

A girl, aged 16 months at the first admission, was born spontaneously at term and was fed on dried milk. At the age of 6 weeks she developed infantile eczema, and at 6 months the introduction of egg into her diet resulted in vomiting and urticaria, which recurred whenever eggs were given. She had no other illnesses until 14 months, and there was no family history of allergy.

At 14 months she began to have attacks of abdominal pain and vomiting, lasting for a day or two at a time, and her appetite became poor. She passed normal stools between and during the attacks. On admission to Maryfield Hospital, Dundee, at 16 months of age, she was found to be a well-nourished child with extensive eczema, involving principally the shoulders, chest, and knees. No other abnormality was found and no mass was palpable in the abdomen. The haemoglobin was 11 g.%, the leucocyte count 12,000 per c.mm., and the erythrocyte sedimentation rate was 40 mm. in the first hour (Westergren). The urine was normal. There was mucus and a little red blood in the stools, culture of which showed no pathogenic organisms. The Mantoux test was positive, but *x*-ray examination of the chest showed only increased broncho-vascular markings.

While in the ward she was inadvertently given an egg custard and had an immediate reaction consisting of generalized urticaria, vomiting, and abdominal pain, which promptly subsided on treatment with an antihistaminic drug.

A provisional diagnosis of gastro-intestinal allergy was made, but a barium enema examination was carried out to exclude any other condition in the bowel. There was a temporary hold-up of the barium in the ascending colon, but eventually normal filling was obtained. The possibility of an intussusception which had been reduced by the enema was considered, but as the symptoms had subsided she was allowed to go home.

She had no more trouble until two months later, when she was readmitted because of further attacks of abdominal discomfort followed by the passage of blood in the stools. Apart from extensive eczema, no abnormality was found on physical examination. A second barium enema showed good filling of the colon but defective filling of the caecum. Gastro-intestinal allergy was still thought to be the most likely cause of her symptoms, and as she was thriving she was discharged from hospital.

One month later she had three more attacks of abdominal colic with blood in the stools, and this time a horizontal thickening was palpable in the upper abdomen. At laparotomy (Mr. W. G. Campbell) a basal appendicular intussusception was found which had further intussuscepted into the colon. The intussusception was easily reduced except for the part formed by the base of the appendix, and the fundus of the caecum with the appendix was removed. Microscopical examination showed diffuse post-inflammatory fibrosis in the subserosa of the appendix, while the mucosa

and submucosa of the caecum showed diffuse and heavy infiltration with eosinophil leucocytes. Post-operative progress was satisfactory, and one year later she was very well, although the eczema was still present.

Discussion

The outstanding symptom of intussusception of the appendix is intermittent abdominal pain, often accompanied by nausea and vomiting, and the stools are usually normal (McSwain, 1941). Many symptoms have been attributed to gastro-intestinal allergy, but intermittent abdominal pain and vomiting are those most commonly mentioned. Blood in the stools is often described as a feature of gastro-intestinal allergy, especially in infants, while it is usually scanty or absent in chronic intussusception (Gaymer Jones, 1924). The difficulties of proving that gastro-intestinal symptoms are allergic have been discussed by Ingelfinger, Lowell, and Franklin (1949), who concluded that absolute confirmation is virtually impossible. They considered such a diagnosis reasonable "if gastro-intestinal symptoms invariably follow contact with a normally inactive substance, and if other causes such as psychic effects and mechanical irritation are excluded." The history of sensitivity to egg of the second patient, and the vomiting and urticaria produced by its administration, favoured the diagnosis of gastro-intestinal allergy, and it seemed reasonable, in the absence of other demonstrable cause, to ascribe the abdominal pain and blood in the stools to allergy as well. Beaven (1929) reported a somewhat similar case, his patient being a girl of 3 years who had florid eczema, and who developed urticaria if she ate eggs. The child had had peri-umbilical pain for one month before laparotomy was carried out. In this child, however, an intussusception was found involving a Meckel diverticulum.

In the present Case 1 there was less justification for attributing the symptoms to allergy, but the history of eczema, the absence of abnormal signs in the abdomen, and the duration of symptoms were misleading. The initial diagnosis was not sustained after admission and the true cause became apparent when the abdominal mass was felt.

Intussusception of the appendix was first recorded in a boy aged 7 (McKidd, 1859), and the majority of cases since reported have occurred in the first decade of life (Forshall, 1953). The condition is usually chronic, the average duration of symptoms before operation being eight months in the 77 cases reviewed by McSwain (1941). Although all cases may not arise in the same way, it is thought that the condition is usually due to peristaltic contractions of the appendix, initiated by attempts to expel a foreign body or by a local thickening of the appendix itself (Fraser, 1943).

It seems possible that in the present cases increased peristalsis resulting from allergic reactions facilitated the occurrence of intussusception, although the eosinophilia in the caecal wall in the second case cannot be adduced as evidence, because the significance of such infiltration is not clearly understood. Moreover, since neither McSwain (1941) nor Fraser (1943) mentions allergy in reviewing the literature on intussusception of the appendix, it appears unlikely to be a common cause, and the association may have been fortuitous.

Summary

Two cases of chronic intussusception of the appendix in young children are reported.

The symptoms were at first attributed to allergic reactions because of a history of infantile eczema in the first child and the presence of infantile eczema and gastro-intestinal allergy in the second.

I am grateful to Professor J. L. Henderson for permission to report these cases, and to Dr. Kenneth Rhaney for the pathological report.

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SURGERY IN OCTOGENARIANS

BY

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The problems caused by lesions in aged patients which constitute either an immediate or a delayed threat to life require careful consideration if an adequate solution of them is to be found. It is recognized that the number of these patients in this country is gradually increasing, and these matters will assume greater proportions in the years to come. A study of a personal series of 34 octogenarians who underwent surgical operation demonstrates that much can be done to help these aged patients even though they are afflicted with the worst forms of malignant disease. The patients were divided into two groups: with malignant and with non-malignant lesions. Their details are set out in Tables I and II.

There were 24 patients with malignant disease; for the majority a major operation was required, and four died. The follow-up of the survivors is shown and six patients were still alive up to twenty-nine months after the operation. Three patients died of non-malignant causes 3 years, 4¼ years, and 4 years 5 months later. Of those who died of cancer four lived for a minimum period of two years.

Distribution and Management of the Lesions

In 15 patients a carcinoma of the alimentary tract was present, and in 9 it was situated in the colon or rectum. One patient suddenly developed a perforation of the pelvic colon proximal to an annular constricting carcinoma. Another suffered from complete intestinal occlusion caused by carcinoma of the pelvic colon.

For an aged patient with an obstructing carcinoma of the transverse or pelvic colon Paul's operation is a satisfactory procedure, provided any involved regional lymph nodes can be removed at the same time. When the affected loop of bowel cannot be exteriorized—for example, in the lower part of the pelvic colon, or at the recto-sigmoid junction—Hartman's operation is useful. For non-obstructing lesions of the transverse or distal colon a partial colectomy with an end-to-end anastomosis is usually radical enough rather than a more extensive left hemicolectomy. When the carcinoma affects the right half of the colon a right hemicolectomy in one stage is advisable if there is no obstruction.

In one patient a large pelvic abscess complicated a carcinoma of the pelvic colon and caused severe toxaemia. A series of staged operations were performed, including drainage of the abscess, the institution of a defunctioning transverse colostomy, partial colectomy, and closure of the colostomy later. The timing of these staged operations is important, so that the patient's general condition is at its optimum when the next stage is performed. By this means many aged patients, even when seriously ill, can be brought through safely. It is worth noting that a palliative colostomy for a carcinoma of the rectum in two patients was followed by survival periods of 2½ and 3½ years. Another patient lived 4½ years after a perineal excision of the

rectum and died of another cause at the age of 87. In some aged patients the application of coagulation diathermy to an obstructing carcinoma of the rectum is a useful method of treatment; one patient lived 2 1/12 years.

TABLE I.—*Octogenarians with Malignant Lesions Treated Surgically*

Age of Patient	Disease	Operation	Immediate Result	Late Result
85	Carcinoma rectum	Left inguinal colostomy	Recovered	Died 2½ years later
88	,, ,,	,, ,,	,,	Died 3½ years later
83	,, ,,	Left inguinal colostomy and perineal excision of rectum	,,	Died 4½ years later of another cause
90	,, ,,	Diathermy to growth	,,	Died 2 1/12 years later
83	Carcinoma colon, perforated	Partial colectomy; later prostatectomy	,,	Died 1 year after first operation
	Enlarged prostate; retention of urine			
83	Carcinoma colon —complete intestinal occlusion	Partial colectomy, Paul's operation	,,	Alive 2 years later
82	Carcinoma pelvic colon	Partial colectomy, Hartman's operation	Died	—
81	Carcinoma pelvic colon with pelvic abscess	Drainage of abscess, defunctioning transverse colostomy, partial colectomy, closure of colostomy	Recovered	Alive 9 months later
86	Carcinoma transverse colon	Partial colectomy	,,	Alive 1 year later
81	Carcinoma caecum	Right hemicolectomy	,,	Alive 9 months later
80	,, stomach	Subtotal gastrectomy	,,	Died 7 months later
80	,, ,,	Gastro-jejunostomy	,,	Died 8 months later
81	Carcinoma oesophagus	Partial oesophagogastrectomy	Died 7 weeks after operation	
80	,, ,,	,, ,,	Recovered	Died 22 mths later
81	Sarcoma breast	Irradiation and radical mastectomy	,,	Died 3 months later
82	Carcinoma breast	Irradiation and mastectomy	,,	Died some months later
80	,, ,,	,, ,,	,,	Died 3 years later
81	Carcinoma tongue (recurrent)	Partial glossectomy	,,	Alive 7 months later
89	Carcinoma floor of mouth and mandible	Partial excision of floor of mouth and mandible. Partial block dissection of neck	,,	Alive 2 5 12 years later
82	Carcinoma prostate. Diabetes mellitus	Suprapubic cystostomy	Died	—
87	Carcinoma pinna	Excision	Recovered	Died 3 years later of another cause
81	Rodent ulcer of face	,,	,,	Lost sight of later
82	Rodent ulcer of orbit	Exenteration of orbit	,,	Died 4 5 12 years later of another cause
83	Vesical calculus and carcinoma of prostate with retention of urine	Lithotrity and transurethral resection of prostate	Died	—

TABLE II.—*Octogenarians with Non-malignant Lesions Treated Surgically*

Age of Patient	Disease	Operation	Immediate Result
86	Rectal prolapse	Thiersch's operation	Recovered
83	,, ,,	,, ,,	,,
80	Enlarged prostate	Prostatectomy	,,
84	,, ,,	,,	,,
80	Crohn's disease of ileum	Enteroenterostomy	,,
80	Chronic cholecystitis	Cholecystectomy	,,
85	Strangulated femoral hernia	Herniotomy	,,
81	Ulcerated angioma of breast	Mastectomy	,,
80	Senile tuberculosis of shoulder-joint	Sequestrectomy	,,
84	Necrotic ulcer of pinna	Excision of pinna	,,

The re-establishment of normal micturition in aged patients who have undergone an operation for carcinoma of the pelvic colon or rectum is often a difficult problem to solve. In males this is often caused by a benign enlargement of the prostate, and surgical treatment is necessary. When the prostate is small and fibrous a transurethral resection is satisfactory, but if the gland is large and fleshy it is probably better to enucleate it, provided the patient will stand the operation. Retention of urine in females can be very troublesome, and regular catheterization is required until normal micturition is re-established. This was necessary for several weeks in the case of the patient with the pelvic abscess complicating a carcinoma of the pelvic colon.

Two patients with a carcinoma of the oesophagus underwent a partial oesophago-gastrectomy; one died in hospital seven weeks later, the other lived for twenty-two months. The problems of each patient with a carcinoma of the oesophagus must be considered carefully, and if possible normal deglutition should be restored. When radical surgical treatment is contraindicated, the insertion of a Souttar tube or a course of high-voltage *x*-radiation often helps. A palliative gastrostomy is an unsatisfactory procedure.

One patient with a recurrent carcinoma of the tongue had a partial glossectomy and was well seven months later; the original lesion had been treated by a partial glossectomy and block dissection of the neck seven years previously. Another patient had a large carcinoma involving the floor of the mouth, mandible, and regional lymph nodes, and complete surgical excision gave a good result; the patient was well 29 months later. In these cases no attempt is made to reconstruct the mandible, since it is better to accept the deformity rather than submit the patient to a further operation and thus prolong his stay in hospital.

Ten patients with non-malignant lesions underwent operations, and all recovered (Table II).

Rectal prolapse is frequent in aged patients, and causes much suffering. Thiersch's operation is valuable because it is simple and usually provides complete control of the prolapse. For example, the patient who was aged 86 when operated on was at the time of writing 90 and never ceased to be grateful. In some patients the silver wire breaks after a time, but it can be easily removed and a new one inserted. Prostatic obstruction is another common condition in aged patients; each must be carefully considered and the appropriate treatment carried out. Two patients underwent prostatectomy with uneventful recovery.

General Considerations Regarding Surgery in the Aged

The health of these patients varies greatly, and it is necessary to assess their biological rather than their chronological years. Some tissues age more quickly than others, and the processes of repair may be retarded considerably in certain parts of the body. Repair is, of course, slower and less complete in old than it is in young tissues, and this fact should be remembered in this branch of surgery. Degenerative changes in vital organs are frequent and cause complications after operations: the most serious and frequent are those of the cardiovascular and renal systems. As a principle it is advisable to perform the simplest operation designed to cure the particular condition causing symptoms. In aged patients nutritional defects are common owing to an inadequate dietary intake, errors in food assimilation, derangement of water and electrolyte balance, and vitamin lack. These factors combine to interfere with wound-healing, lower the resistance of tissues to infection, and generally decrease the patient's power to withstand a major surgical operation.

It is advantageous to have a clear picture of the changes often present in many organs in aged patients, and reference is made to the work of Howell and Piggot (1951) regarding the morbid anatomy of various systems of the body. Important findings included the following: considerable atheroma was present in the aorta in 68% of patients, in the coronary arteries in 36%, and in the cerebral arteries

in 25%. Changes in the heart were patchy myocardial fibrosis in 44%, old coronary infarcts in 12%, fatty myocardial changes in 12%, and brown atrophy in 20%. Amongst the changes in the respiratory system were atrophic emphysema in 64%, hypertrophic emphysema in 28%, pulmonary oedema in 68%, bronchopneumonia in 32%, pleural effusion in 12%, and chronic bronchitis in 32%. Normal kidneys were found in only 12% of patients; the pathological changes in others included coarse scarring, granular surface, narrow cortex, thickened vessels, and excess fat.

The common post-operative complications according to their frequency and potential threat to the patient's life are the following. Bronchopneumonia is frequent and serious and often diffuse, with few demonstrable chest signs. Thus cough is slight but there is fever, and radiological changes in the chest are common. Cardiac complications are frequent and include congestive heart failure, coronary occlusion, and various arrhythmias. Pulmonary oedema is a frequent accompaniment of heart complications. Peritonitis is an important complication of various intra-abdominal lesions and operations; this is not tolerated well by aged patients. Attention is called to the frequency of infections in the urinary system which may be serious, and therefore early recognition and treatment is essential. Post-operative thrombosis and embolism often occur, and fatal embolism may be an accompaniment of impairment of the heart's action. Mental disorientation is worthy of special mention; the strength of the patient's mentality to withstand a major operation must always be assessed beforehand.

Special Points Regarding Treatment

Provided intelligent management is instituted many aged patients are brought safely through major surgical operations. Obviously emergency surgery is more risky than elective surgery, and according to Carp (1951) the former carries a mortality rate about two and a quarter times greater than for the latter. An adequate time is allotted for pre-operative treatment when elective surgery is contemplated, and nutritional defects are rectified as much as possible by routine methods. Oral feeding is preferable to other routes whenever this can be done, and a high calorie diet rich in vitamins is given. Any disturbance in the water and electrolyte balance is rectified, and when parenteral fluids are necessary they are selected carefully and administered according to the patient's exact requirements, controlled by laboratory investigations. Physiotherapy is given routinely, and the importance of the correct psychological approach is mentioned because aged patients require tactful and kindly handling in an optimistic atmosphere.

During the Operation.—Attention is given to the usual details of operative technique. Exaggerated positions on the table and rapid changes in the position of the patient are avoided. Any undue exposure of tissues is eliminated and careful haemostasis is essential. The circulatory system must not be overloaded with intravenous fluids, including blood, and the amount given of the latter is assessed by measuring the blood loss during the operation.

Anaesthesia

Premedication is minimal in amount and controlled carefully, because respiratory depression is easily induced. Therefore small doses of "omnopon," ½ to ¼ gr. (10 to 20 mg.), or pethidine, 100 mg. or less, are given; the exact dose chosen is related to the body weight, general condition, and the results of the liver- and renal-function tests. Anaesthetic induction is smooth and adequate oxygenation is essential. It is necessary to avoid deep anaesthesia and marked fluctuations in the blood pressure. For the usual abdominal operations the following is a satisfactory method. The patient is anaesthetized with intravenous thiopentone sodium and not more than 0.5 g. is given. An endotracheal tube is passed and anaesthesia is continued with pethidine, given intravenously (dose 50 mg.); this is repeated if necessary, but not more than 25 mg. is given. For abdominal relaxation tubarine up to 15 mg. is sufficient. A clear airway must be maintained, and bronchial aspiration may be required during and after the operation.

Post-operative Care

Routine methods are employed and emphasis is placed on certain aspects. Fluid, including blood, and electrolyte requirements are assessed and provided carefully. The manifestations of potassium deficiency, acidosis, or alkalosis are recognized early in their development and treated. Oral feeding is resumed as soon as possible. For post-operative sedation barbiturates are avoided and reliance is placed upon small doses of the morphine derivatives; chloral and bromides are also useful. Physiotherapy is given routinely, and early ambulation is required unless contraindications are present.

Summary

A series of 34 octogenarians who underwent surgical operation is reported. They are divided into two categories—those with malignant and those with non-malignant lesions—and the end-results are given. Certain principles in their treatment are stressed, and it is shown that many of them can be brought safely through major surgical operations.

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Medical Memoranda

Recovery after Overdosage with Methylpentynol

Methylpentynol is now being prescribed fairly widely as a hypnotic and as a means of allaying apprehension, but there are relatively few reports of the results of overdosage. The following case report describes recovery after ingestion of approximately 10 g. of methylpentynol in the form of a proprietary elixir ("oblivon"), and this appears to be the largest recorded overdosage followed by recovery.

CASE REPORT

A woman aged 39, weight 7 st. 8 lb. (48.3 kg.), was admitted to hospital on October 29, 1954, having taken approximately 5 oz. (150 ml.) of a proprietary elixir containing 250 mg. of methylpentynol per drachm (70 mg. per ml.). There was a history of previous mental illness, necessitating admission on a voluntary basis to a mental hospital in July, 1954, for a period of ten days. Since that time the patient had been depressed at intervals and had threatened to commit suicide.

About mid-morning on October 29 she drank 5 oz. (150 ml.) of methylpentynol elixir and was admitted to hospital at 3.30 p.m. She was then deeply asleep, but responded faintly to pressure on the supraorbital nerve. Her general condition was good: respirations 20, pulse 72, blood pressure 130/80. Her pupils were normal in size and reacted briskly to light.

On the assumption that absorption was probably complete, stomach lavage was not carried out. General supportive treatment was instituted, and methylamphetamine hydrochloride, 30 mg., was given intravenously every half-hour. At 6.30 p.m. she was less deeply asleep, and half opened her eyes when spoken to. Her general condition was good: respirations 21, pulse 72. By 8.30 p.m. she was half awake and making attempts to speak. At 10 p.m. she was able to speak and to answer questions, although still drowsy. At this point the methylamphetamine hydrochloride was discontinued.

During the next day she was somewhat drowsy, but rational and capable of answering questions. By the morning of October 31 she was fully recovered and later consented to have further mental hospital treatment as a voluntary patient.

Animal experiments conducted by Margolin *et al.* (1951) showed that the dose that killed 50% of test rodents (LD_{50}) varied from 600 to 900 mg. per kg. Recoveries in adult humans have been reported after ingestion of 6.25 g. (Bourne, 1954) and 8.75 g. (Lemere, 1952), but the single fatality on record is thought to have followed the ingestion of 4.5 to 6 g. (Cares *et al.*, 1953).

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Case of Fallot's Tetralogy with Multiple Congenital Abnormalities

The past decade has shown rapid advances in the understanding of congenital heart disease. The work of Blalock and Taussig (1945), Taussig (1948), and others has resulted in major progress in the surgical treatment of the three main conditions: pulmonary stenosis, patent ductus arteriosus, and coarctation of the aorta. An increasing number of other congenital conditions associated with morbus caeruleus are coming gradually to light. Thus, Campbell and Deuchar (1953), in a series of 200 cases of morbus caeruleus, found two cases of dextrocardia with complete situs inversus, one with isolated dextrocardia, and three with laevocardia plus transposition of abdominal viscera.

CASE REPORT

On February 10, 1953, an Indian boy aged 8 was brought to hospital. A typical history of a "blue baby" who had survived repeated attacks of endocarditis was elicited from his father. He had always been delicate and easily got dyspnoeic on moderate exertion. He was, however, able to attend school, where he had a good record. He could not take part in any games. He suffered frequently from attacks of cough, associated with high fever, and had some difficulty in swallowing. His appetite was poor. He had also suffered from abdominal discomfort associated with constipation. His present history was that of fever, cough, and severe dyspnoea, lasting for three days.

Clinical Findings.—He was rather thin and proportionally built, but somewhat small for his age. He walked into the examination room unaided with some effort. There were cyanosis of moderate degree and marked clubbing of fingers and toes; temperature 102° F. (38.9° C.), pulse 124, respirations 36; marked dyspnoea; multiple bilateral rhonchi in lungs. Heart: forceful, visible apex beat in fifth left interspace; a loud diffuse systolic murmur was heard along the left sternal border, maximal over the third and fourth left interspace, but audible also over a wide area on the front and back of the chest. Abdomen: soft, not tender, some fullness below umbilicus; spleen and liver not palpable. Rectal examination: hard faecal masses in ampulla.

A provisional diagnosis of pulmonary stenosis, probably a Fallot's tetralogy, complicated by infective endocarditis, was made. The father of the boy refused to have him admitted. Blood cultures could not be taken during the acute phase of the illness. Treatment and subsequent investigations had to be carried out in the out-patient department.

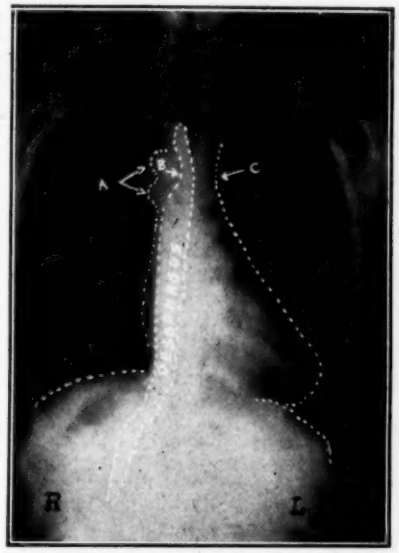
The acute symptoms subsided after a week of massive penicillin therapy and rest, making the following investigations possible.

X-ray films of the chest revealed a boot-shaped heart of normal size; replacement of aortic knuckle and pulmonary convexity on left upper cardiac margin by a concavity; dextroposition of aorta, being visible in two distinct cones within shadow of superior vena cava; marked passive pulmonary hyperaemia.

Blood picture: Polycythaemia, with Hb 108%; erythrocytes, 6,910,000; leucocytes, 9,400 (polymorphs 48%, lymphocytes 44%, monocytes 1%, eosinophils 7%).

Barium swallow examination was done to ascertain the relationship of the oesophagus to the great vessels (see Fig.).

Diagnosis.—These clinical, radiological, and haematological findings strongly suggested a diagnosis of Fallot's tetralogy—that is, pulmonary stenosis; dextraposition of aorta with widening, dextraseptal deviation, causing aorta to override septum; high ventricular septal defect resulting in a persistent venous-arterial shunt; right ventricular hypertrophy (Boyd, 1947). According to Mercer (1950), this dextraposition of the aorta is present in 25% of all cases of Fallot's tetralogy.



Barium swallow examination in Fallot's tetralogy: boot-shaped cardiac silhouette with concavity (C) at left upper margin; dextroposition of aorta seen in two cones (A); upper oesophagus lying to left of great vessels, being indented markedly on right margin by aorta (B); lower oesophagus deviating to right, indicating dextrogastria.

In view of the dextrogastria, a complete radiological investigation of the gastro-intestinal tract was then carried out.

Barium meal examination revealed a situs partialis inversus: dextrogastria; inversion of duodenum; position of duodeno-jejunal flexure not clearly seen; terminal ileum, caecum, appendix, and colon appear again in normal position. The appendix seems to have a rather wide lumen. In the left half of the transverse colon an area of early diverticulosis was discovered, extending as far as the splenic flexure. The affected segment showed fine spastic notching on the summits of the haustral bulges, described first by Spriggs (1929) to be characteristic of early diverticulosis. Barium-filled diverticula could be seen in the same segment after partial evacuation and after complete evacuation of the barium meal. The latter, as well as a subsequent barium enema, showed a megarectum. Sigmoid and rest of colon were of normal calibre.

Lastly, congenital absence of the left twelfth rib was found as a curious skeletal abnormality.

COMMENT

The pulmonary stenosis in this case may not be very marked, as cyanosis was moderate and the degree of exercise-tolerance fair, even after several previous attacks of infective endocarditis. The abnormal findings in the gastro-intestinal tract may well explain the abdominal symptoms of this patient, bearing in mind that children with morbus caeruleus often suffer from digestive disturbances and put on weight rather slowly.

It may serve as a good example that in congenital heart disease multiple extracardial congenital abnormalities can be expected, particularly within the thorax and abdomen.

A deliberate search for such abnormalities is advisable before operative measures, such as creation of an artificial ductus arteriosus or pulmonary valvulotomy, are considered, as it has been found (Campbell and Deuchar, 1953) that the mortality in cases with complex lesions is rather high.

I wish to thank the Director of Medical Services, Federation of Malaya, for permission to publish this case. I am also indebted to the staff of the *x*-ray department of the Malacca General Hospital for the radiograph, and to Dr. M. Rajathurai, of the pathology department, for the blood studies.

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Nervous Manifestations of Infectious Hepatitis

Neurological complications of infectious hepatitis are rare (Cameron, 1943). The incidence is varied—for example, 2 in 170 cases (Harries and Mitman, 1951), 3% (Lucké and Mallory, 1946), and 32 in 151 (Hardy and Feemster, 1946).

CASE 1

A girl of 6 years vomited once on January 7, 1954, but was well until the 11th, when vomiting recurred with lethargy.

She was admitted to hospital on the 12th with severe lethargy and general hypotonia. *X*-ray films of chest and skull were normal. The next day there was rapid deterioration. She was grey, limp, and incontinent of urine, and fluids were regurgitated through the nose. Light coma; fundi normal. She was given an intragastric drip and postural drainage with suction. In the evening there was generalized spasticity.

Laboratory Findings.—C.S.F. normal. Blood urea, 40 mg./100 ml.; blood sugar, 60 mg./100 ml.; alkali reserve, 59.5 vols. CO_2/100 vols.; W.B.C., 13,000 c.mm. (polys. 84%, lymphs, 9%, monos. 7%). Serum protein: total 7 g./100 ml. (albumin 4.4 g., globulin 2.6 g.). Serum alkaline phosphatase, 16 units; thymol turbidity, 5 units; thymol flocculation, ++. Serum did not appear jaundiced.

On January 14 there were frequent vomits of altered blood; marked dehydration; slight scleral icterus. T. 102.8° F. (39.3° C.); P. 144; R. 40. I.V. infusion started. Crystalline penicillin, ¼ mega unit i.m. 6-hourly. Vitamins K and B complex. Deep coma; no paralysis; spastic limbs. Extensor plantar and absent abdominal reflexes. Second lumbar puncture still normal, but pressure 200 mm. Trace of bilirubinuria. January 15: Still intense coma; vomiting altered blood; cyanosed; noisy bubbling respiration; flaccid quadriplegia and extensor plantar response. January 16: Rational; jaundiced; liver not enlarged. January 18: Still incontinent of urine and faeces. I.V. drip discontinued. W.B.C., 23,000 per c.mm. (polys. 93%, lymphs. 4%, monos. 3%). January 23: Jaundice fading. January 29: C.N.S., normal; no icterus; liver normal size. Virus studies and leptospiral agglutination: negative. March 10: Discharged from hospital. June 8, 1954: Child physically fit, but irritable, with no sequelae. Liver function tests, including the zinc sulphate turbidity for γ-globulin (6 units), were all normal.

CASE 2

A girl of 9 years was admitted to hospital on April 5, 1953, with a week's history of vomiting, headache, and severe lethargy. Three siblings had jaundice. She was very drowsy, with slight scleral icterus; liver tender and enlarged three fingerbreadths. Temp. 96° F. (35.6° C.), later 100.4° F. (38° C.). Dehydrated. Given adequate i.v. fluids with vitamin K i.m. Retention of urine until April 7, when she had severe lethargy and bilirubinuria. April 8: Conscious; well jaundiced, with liver enlarged. I.V. fluids discontinued.

Laboratory Findings.—Blood urea, 34 mg. per 100 ml.; W.B.C., 7,400/c.mm. (polys. 54%, lymphs. 42%, monos. 3%, basos. 1%). Alkaline phosphatase, 22.5 units; thymol turbidity, 14 units; thymol flocculation +++. Total serum protein, 5.5 g./ml. (albumin 3.0, globulin 2.5). Some degree of liver dysfunction present.

The patient was discharged from hospital on May 29. When seen on June 8 she was symptom-free, having no nervous sequelae except slight irritability. Liver palpable one fingerbreadth. Liver function tests normal, except zinc sulphate turbidity for γ-globulin (11.6 units), the local normal range being 2.8 to 10.4 units.

PATHOLOGY AND PATHOGENESIS

Briefly, there is an acute non-specific degeneration of ganglion cells with some oedema of the brain without demyelination and, in 15% of fatal cases, a mild meningoencephalitis (Lucké, 1944). Apart from any direct action of virus upon the central nervous system, loss of the detoxifying function of the liver may account for the nervous manifestations.

Walsh (1951, 1953) suggests that hepatic coma may be due to inability of the brain cell to combine glutamic acid with ammonia to form glutamine (Weil-Malherbe, 1950), whilst Riddell and McDermott (1954) postulate the coma in chronic liver disease as due to ammonia intoxication.

DIAGNOSIS

Nervous manifestations may take the form of encephalomyeloradiculitis, with both motor and sensory involvement (Zimmerman and Lowry, 1947). On the other hand, the signs may be encephalitic with or without motor paralysis or post-infectious sequelae. Clinically, the case may resemble uraemic or diabetic coma. Coma caused by other infections and narcotic or toxic poisoning must be considered; so must psychic states.

TREATMENT

There is no specific remedy. Davidson and his colleagues (Gabuzda *et al.*, 1952; Phillips *et al.*, 1952) note the potential danger of a high-protein diet and of ammonium-containing compounds in decompensated liver disease, but stress the therapeutic value of protein in compensated liver disease. Glutamic acid therapy in hepatic coma is not yet of proved value (Riddell and McDermott, 1954). Hypertonic glucose or double-strength plasma (i.v.) for cerebral oedema, paraldehyde for excitement, and antibiotics for secondary infection may all be helpful. Bed rest should be maintained until there is no bilirubinuria and the liver is no longer enlarged.

There are three points I would like to stress: (1) The nervous manifestations preceded the jaundice and cleared up as it became marked. (2) The course may be reversible, and recovery may occur after apparent hopelessness. (3) This infection may be the cause of some cases of idiopathic encephalitis or meningoencephalomyelitis.

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Reviews

ANY QUESTIONS?

Any Questions? A Selection of Questions and Answers published in the *British Medical Journal*. Third series. (Pp. 227 +xv. 7s. 6d.) London: British Medical Association. 1954.

The third series of *Any Questions?* is as informative and as entertaining as ever, and there seems no end to the usefulness of this feature of the *Journal*. One of the advantages or disadvantages of being a medical don is that one is so often asked at high table the sort of question the answer to which is not provided in the medicine one teaches to students, and one therefore sympathizes with the inquiries which have given rise to these books. Bizarre questions of this type in the present volume include, "Will a wig stop hair growing?" (answered by the single word, "No"), and the results of intermarriage of white and black, answered at much greater length and with the aid of a funny story. A second group of questions concerns new knowledge which has not yet reached the textbooks, as, for example, the best treatment of amoebiasis, the danger of vaccination in pregnancy, and the carpal tunnel syndrome. Then come questions which are off the normal beat of the practitioner—sanitation and hygiene, the sound-proofing of surgeries, and the prevention of scale in sterilizers. Finally, there are the marital, sexual, and psychological problems, which begin to shade into the world of Dorothy Dix. The answers are fair game for the medical sharpshooter. I should strongly disagree with the view that alcohol is not a diuretic, which is contrary to the experience of William Shakespeare (*Macbeth*, Act II, Scene III, line 27) and the experiments of Maurice B. Strauss.

This third volume is provided with a cumulative index which makes it more than ever apparent that a series of this kind gains strength by growth in size and that it will eventually become a valuable encyclopaedia of medicine.

L. J. WITTS.

BIOCHEMISTRY FOR THE CLINICIAN

Biochemical Investigations in Diagnosis and Treatment. By John D. N. Nabarro, M.D., M.R.C.P. (Pp. 299+ix. 25s.) London: H. K. Lewis and Co., Ltd. 1954.

Dr. Nabarro has covered a wide field in these 300 pages and is to be congratulated on providing a very readable account of the subject. He has avoided all matters of technique, and has not devoted much space to the underlying biochemical mechanisms. The book, therefore, is mainly a guide to the clinician on what tests may usefully be carried out on patients with metabolic diseases and on how he should interpret the results. The author's purpose is much enhanced by the rather original index, which is in two parts: "Index of Investigations" and "Index of Conditions, Diseases, and Treatment." This will be a great help to those using the book. The actual arrangement of chapters is a compromise between the two, some being devoted mainly to diseases, and others mainly to tests or types of biochemical derangement.

The text is well written, up-to-date, and remarkably condensed. It is inevitable from its size that certain sections should appear to be rather too brief; for example, the sections on renal tubular acidosis and on hypoglycaemia could be expanded with advantage. The arrangement of references is somewhat puzzling. A few key references are found at the end of certain chapters, and in addition 91 more are given at the end of the volume, but the subject to which each of them refers is not indicated. The usual table of normal values at the end is comprehensive and valuable. This book will undoubtedly be useful to the busy clinician who wants a quick reference to possible biochemical disturbances in his patients, and equally to the laboratory worker faced with an unusual type of biochemical disturbance.

N. F. MACLAGAN.

ANAESTHESIA IN GERMANY

Die Narkose, Ein Lehr- und Handbuch. By Professor Dr. H. Killian and Professor Dr. H. Weese. (Pp. 1,003+xxviii; illustrated. D.M. 170.) Stuttgart: Georg Thieme. 1954.

The massive, all-inclusive tome, of which this is an impressive example, is becoming increasingly rare among medical books. Few men feel able to write with authority on more than one or two facets of their work. Most are specialists even within their specialties. When teaching they must be careful not to assume either that the other man's field is easier than their own, and thus treat it superficially, or that it is specially difficult and so ignore it altogether or labour too painfully to expound it. Both these mistakes are made in this book. The title suggests that the clinical administration of anaesthesia would form the book's backbone. The authors, however, are not anaesthetists themselves, and though some (but only some) of the appropriate sections have been rightly delegated to their anaesthetic colleagues Drs. Irmer and Koss, the general balance of subject-matter does not seem correct. The sections dealing with the administration of anaesthesia are too short and the rest are too long. In a textbook of anaesthetics detailed accounts of pharmacology, pathology, physiology, and so forth are acceptable in their proper proportions and when relevant to the main subject-matter. The impression given here is that they are almost ends in themselves.

It is doubtful whether this book will help in a practical way the young anaesthetists in training in Germany. They need something more akin to our own *Essentials of General Anaesthesia* by Macintosh and Bannister. They might conceivably get the impression that a detailed theoretical knowledge of such related sciences as pharmacology, physiology, pathology, and haematology would compensate for their inability to pass a tracheal tube or to distinguish between respiratory obstruction and overdose, or for their lack of long experience in the operating theatre. Teachers and experienced anaesthetists in general, however, will regard the book differently: they will appreciate the enormous field from which the material has been collected and admire the herculean labours of compiling it. They will, if their knowledge of German is good, find this massive book valuable as a means of reference to practically everything connected with anaesthesia, even if sometimes only distantly related.

The publishers have done an expert job, and the volume is a pleasure to handle and to look at. Lavish reference lists are given at the end of each subject and an adequate index is included. A sad note is struck by an obituary notice of Professor H. Weese, one of the co-authors, who did not live to see the book in print.

W. W. MUSHIN.

THE FIRST ELIZABETH'S HEALTH

Elizabeth Tudor: The Lonely Queen. By Sir Arthur Salusbury MacNalty, K.C.B., M.A., M.D.(Oxon), F.R.C.P., F.R.C.S. (Pp. 272. 18s.) London: Christopher Johnson. 1954.

Much has been written about the fatal illnesses of monarchs, but their general health has not so often been the subject of comment. Sir Arthur MacNalty, who has made a special study of the Tudor period and two years ago published an interesting study of "Henry the Eighth, a Difficult Patient," has now turned his attention to Henry's talented daughter, Queen Elizabeth the First, and has written an account of this remarkable woman with special reference to her ailments and illnesses. The book is meant for the general reader, who will here find in small compass a readable account of this exciting period of English history. Medical readers will turn with special interest to Chapter XV, where is discussed "The Queen's Medical Case-book."

Queen Elizabeth was for the most part a strong woman, both physically and mentally, and we find it difficult to accept the view that she was hysterical. She successfully overcame such serious diseases as smallpox and nephritis, and continued hunting and dancing up to the last year, the seventieth, of her life. She appreciated the advice of her

physicians, but did not always follow it, for when on one occasion she was ill of what was thought to be food-poisoning she held another opinion of her illness : she thought it was due to the "fact that for three or four years she had found herself so well that she had disregarded all the strict discipline which her physicians formerly had been accustomed to impose upon her by purging her and drawing a lot of her blood from time to time."

In two appendices we are given a list of Elizabeth's suitors and a brief account of her physicians. We wish that space had permitted a fuller account of the latter.

This book is a definite contribution on new lines towards the understanding of a great, complex, and interesting personality, and we believe it will deservedly attract widespread attention.

ZACHARY COPE.

OPERATIVE SURGERY

Textbook of Operative Surgery. By Eric L. Farquharson, M.D., F.R.C.S.Ed., F.R.C.S.Eng. (Pp. 853+viii; illustrated. 75s.) Edinburgh and London: E. and S. Livingstone Ltd. 1954.

Books on operative surgery range from the concise one-volume textbook designed to cover the standard operations for the benefit of a student working for examinations to the large encyclopaedia in several volumes. The first seldom gives enough detail to help the practising surgeon; the second is usually a syndicated work and reflects its multiple authorship in a certain unevenness of standard.

Mr. Farquharson's book is in some ways unusual. First, though in a single volume, it is a large work containing 850 pages; secondly, the whole of it is written by himself, so that it has a uniformity of style and of outlook that makes it easy to read; and, thirdly, it is written with a particular aim and adheres to it throughout. That aim is to afford practical advice to the young general surgeon working for the first time on his own responsibility and unable to call on more expert help.

In pursuing his intention Mr. Farquharson precedes each section with a brief review of the anatomy of the part and of the principles on which operation is based, adding in some sections an account of the use of special instruments such as the cystoscope and the sigmoidoscope. He describes the important and commonly performed operations in considerable detail, with excellent and helpful illustrations, the majority of which are by Miss McLarty. The rarer procedures, or those whose value is more in doubt, he discusses less fully, but in most cases gives references indicating where detailed information can be obtained.

For the purpose for which it is intended this is a really good book, one that should find a permanent place in any surgical library. A London surgeon, even though he be a Scotsman, cannot be expected to agree with all the views of one trained in Edinburgh, but the criticisms that can be offered are of detail only, with one exception. The example of parenteral fluid administration given on page 566—9 litres in 24 hours—is dangerous and should be amended in the next edition.

Messrs. Livingstone have done their job as publishers with their usual craftsmanship.

HENEAGE OGILVIE.

ORTHOPAEDICS OF CHILDHOOD

Kinderorthopädie. By Rupprecht Bernbeck. Introduction by Professor Dr. Med. Georg Hohmann. (Pp. 234; 174 illustrations. D.M. 39.) Stuttgart: Georg Thieme. 1954.

It is inevitable that any volume embracing all the orthopaedics of childhood will be a little uneven. This volume presents the author's personal experience from the German viewpoint. After a general introduction, which is much the best portion of the book, there follow a series of special sections, the largest being devoted to clubfoot and congenital dislocation of the hip. This includes a clear description of the pathological varieties of clubfoot. The treatment is not so clearly described; for example, whereas Kite's plasters are mentioned, there is no reference to the use of Denis Browne's splints. In contrast to these full discussions, the section on slipped femoral epiphysis is short and incomplete, and other sections, on torticollis, osteomyelitis, and the dyschondroplasias, are merely résumés.

The book is well illustrated with radiographs in series, and is worth perusal for these alone. The volume as a whole will appeal to the children's physician, and the fuller sections to the specialist. The book unfortunately evidences the isolation of German medical thought during the past 15 years, from which in other fields, such as virology, it is slowly escaping. In the literature quoted there are surprisingly few references to English and American authors (2 out of 52), and French and Italian authors are given equally scant treatment.

J. G. BONNIN.

THE VITAMINS

The Vitamins: Chemistry, Physiology, Pathology. Volumes I and II. Edited by W. H. Sebrell, jun., and Robert S. Harris. (Volume I: pp. 676+xiii; Volume II: pp. 766+xiii; illustrated. $16.50 each vol.) New York: Academic Press Inc. 1954.

This is an encyclopaedic work on vitamins which is to appear in three volumes, the first two having already been published in the second half of 1954. The treatment is essentially biochemical, although, as the subtitle indicates, chemical, physiological, and pathological aspects are also included. It is not, and is not intended to be, a guide for the general practitioner, or even for the clinical specialist. The manifestations of vitamin deficiency and their therapy are not dealt with in any detail, since, as the authors themselves say, such information is available in other publications. For the same reason methods of vitamin assay are referred to but briefly. The approach is not historical, but rather that of a detailed systematization of existing knowledge in those fields of vitaminology that border on biochemistry. As such it is an invaluable reference work for the laboratory investigator; and the bibliography, which is unusually extensive (although sometimes too exclusively American), will prove extremely helpful.

It is not too much to say that "Sebrell and Harris" immediately becomes an indispensable work of reference for all serious research workers concerned with vitamin science.

LESLIE J. HARRIS.

CLINICAL NEUROLOGY

The Basis of Clinical Neurology. By Samuel Brock, M.D. Third edition. (Pp. 510+xii; illustrated. 54s.) London: Baillière, Tindall and Cox, Ltd. 1953.

In this detailed account of the anatomy and physiology of the nervous system Dr. Brock has been at pains to exclude material not of direct value to the clinical worker. All the same it has taken over five hundred pages, not all lightened by his excellent line diagrams, to say what seems essential. Syndromes are described, but not disease; anatomical dysfunction, and not pathology; so that the student of anatomy will find as much of interest as will the clinical worker. The way function is married to structure makes clinical syndromes easy to understand. For instance, the spinal cord is considered in seven short chapters in which, its anatomy and blood supply having been dealt with, its structure is related to movement, to sensation, and to visceral function; and then it is considered as a conducting and a segmental organ.

Some of the most interesting sections to the reviewer are in small type—which shows the author's discretion—and the arrangement of material enables the student to select with ease. He should not, however, miss the last clinical chapter, on posture, for this is a delightful essay in which the systematic integration of dull facts brings them to life. The closing chapters on the cerebrospinal fluid, radiology, and electromyography are unnecessary in a text of this sort, and do not maintain the standard of the earlier dynamic clinical sections.

DENIS WILLIAMS.

BRITISH MEDICAL JOURNAL
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TREATMENT OF PULMONARY TUBERCULOSIS

The introduction of effective antibacterial drugs has revolutionized the treatment of pulmonary tuberculosis. Whereas in the past the physician treating this disease has had to rely upon the natural resistance of the patient aided by bed rest and by the indirect effect of collapse therapy, he now has available methods of direct attack upon the tubercle bacillus; and the surgeon, previously restrained in the application of some of his more radical methods by the fear of complications, has now much greater freedom of action. These striking changes in the practical management of pulmonary tuberculosis have tended to distract attention from important epidemiological changes which have been occurring more gradually but which may be of greater ultimate importance. As Dr. J. L. Livingstone points out in his Mitchell Lecture, published in the opening pages of this *Journal*, preventive medicine does more to eradicate tuberculosis than the efforts of the clinician in dealing with the sick. This, like most generalizations, needs qualification. It is true without reservation in relation to underdeveloped communities where the pool of infectious cases is too great to be much diminished by the provision of treatment on any practicable scale. But in communities in which good progress has already been made in the control of the disease it seems clear that rigorous clinical measures can make a significant and perhaps decisive contribution towards the removal of the last remaining sources of infection. Dr. Livingstone's main purpose in his lecture was to give an account of his own experience in the management of pulmonary tuberculosis, with special reference to collapse therapy. The measures which he uses include artificial pneumothorax, phrenic paralysis with pneumoperitoneum, and extrafascial apicolysis with "lucite" plombage of the type described by Cleland.[1]

Dr. H. G. Trimble's paper, also published in this issue (p. 250), provides an interesting companion to Dr. Livingstone's. Its approach is entirely different. It describes the analysis of the answers given by one hundred physicians in 1953 to a number of questions about the treatment they would use for ten representative cases of pulmonary tuberculosis which were presented to them in the form of brief case histories and radiographs. The answers are compared with the answers given to the same questions about the same patients in 1948. It is perhaps a valid criticism that the method of presentation of the cases to the physicians was of necessity artificial. It is to be hoped that no physician would seriously give advice about the treatment of a patient on the basis simply of a case history and inspection of a radiograph. Nevertheless Dr. Trimble's analysis gives some indication of change in opinion. It also shows very wide variation in present-day practice. Similar differences of opinion would probably be found in the answers to corresponding questionaries relating to the treatment of a number of other illnesses, especially those where both medical and surgical methods are in use—for instance, peptic ulcer. The most important trend among the American physicians was the greatly increased use of antibacterial treatment. In the far advanced cases every physician would use such treatment, and even in minimal infections only very few would use bed rest without antibacterial treatment. As compared with 1948, artificial pneumothorax showed a remarkable diminution in popularity, while pneumoperitoneum showed a corresponding increase. Dr. Trimble does not state how often the physicians whom he questioned would combine phrenic nerve paralysis with pneumoperitoneum. Dr. Livingstone states that he regards pneumoperitoneum as an adjunct to phrenic paralysis, and that he has never used pneumoperitoneum without phrenic paralysis. This seems to be the logical consequence of the view that collapse therapy must be selective if it is to be effective.

Among the American physicians there has been an increase in the proportion of patients for whom surgical treatment would be advised; and resection has become far more popular than thoracoplasty. Some of the recommendations for resection are surprising; for instance, a few physicians stated that they would advise resection in a case of bilateral acute pulmonary tuberculosis rather more extensive on the left side. Dr. Livingstone makes a useful distinction between resection performed as direct therapy, removing diseased and usually functionless portions of lung which have caused symptoms, and resection performed prophylactically, in which damaged lung not known to have caused symptoms is removed on the ground that it is a potential source of danger at a later date. He adds that in some countries resection after a long course of antibacterial therapy has become the accepted form of treatment. The justification for "prophylactic" resection depends upon the view that residual encapsulated caseous foci

[1] *Thorax*, 1950, **5**, 248.
[2] American Trudeau Society, *Amer. Rev. Tuberc.*, 1953, **68**, 477.
[3] Goodwin, R. A., *Ann. intern. Med.*, 1954, **41**, 328.
[4] Foster-Carter, A. F., *et al.*, *Brompton Hosp. Rep.*, 1952, **21**, 1.

are dangerous; but no agreed opinion about how dangerous they are has yet been reached.[2]

One of the most remarkable changes which has occurred since the advent of antibacterial treatment has been the great diminution in the popularity of artificial pneumothorax. Some physicians have ceased to use this form of collapse therapy.[3] Dr. Livingstone believes that there are still some indications for artificial pneumothorax, and points out that the figures derived from the Frimley follow-up of 1,153 cases, each for a minimum period of eight years, show that even before antibacterial drugs were available a pneumothorax which resulted in closure of the cavity improved the prognosis.[4] The real drawback of artificial pneumothorax treatment was the high incidence of pleural complications. There is, however, reason to believe that the combination of antibacterial drugs with artificial pneumothorax will result in a lower incidence of such complications. No comparably extensive series of cases treated with artificial pneumothorax since the advent of antibacterial drugs has yet been studied. It is to be hoped that Dr. Livingstone and others who continue to use artificial pneumothorax in selected cases will publish their results in due course. It is difficult to believe that a method of treatment which was so widely and successfully used for so long by so many physicians can really have no useful part to play in the future.

In pulmonary tuberculosis to-day the multiplicity of possible therapeutic measures in several fundamentally different categories tends to cause confusion, and the formal assessment of all possible combinations of them is not feasible. But the disease presents such a variety of anatomical and immunological features that it would be surprising if procedures which have proved successful in the past were not found still to be useful in selected cases in combination with antibacterial treatment. In the more usual types of case the physician may hope eventually to have precise data indicating the superiority of one of the possible lines of treatment: in those not falling into a category which has been statistically investigated he will continue to have the responsibility of exercising judgment based upon experience.

HEPATIC FIBROSIS

The idea that nutritional deficiency is of great importance in the causation of liver disease springs mainly from the results of animal experiments performed in the past 15 years.[1 2] These experimental results seemed at first to show why human liver disease is more prevalent in Africa, Asia, and other countries where the standard of nutrition is well below that of Western Europe and the U.S.A. The fatty liver of kwashiorkor in Uganda[3] and of the "fatty liver disease in infants in the British West Indies"[4] were strikingly similar to the fatty livers so readily produced in rats by dietary means and which so consistently caused cirrhosis if maintained for long enough. The failure, however, of methionine or choline to mobilize the fat from the livers of affected children, and the difference in the distribution of the lesions in the human liver from that in the experimental animal, suggested from the first that the direct application of the experimental results to man was altogether too simple. The recent observations of J. H. Walters and J. C. Waterlow[5] on the incidence of hepatic disease in West African children give prominence again to these difficulties, but also help to dispel them.

The clinical material which formed the basis of this study in the Gambia consisted of four groups of children and adolescents up to 20 years of age—a control group of 15, a group of 17 patients with symptomless hepatomegaly, a group of 26 malnourished infants, and a group of 21 children with hepatomegaly associated with oedema and ascites. All cases were studied as hospital in-patients, and the investigations included one or more needle biopsies of the liver in every case. The fibrosis in these biopsy specimens was classified, in increasing order of severity, as normal, periportal quiescent, periportal active, and diffuse. The incidence and severity of the fibrosis in the children with symptomless hepatomegaly were about the same as in the control groups—namely, 50%—and almost entirely of the periportal quiescent type. Walters and Waterlow concluded from this that the hepatomegaly in these children could not be regarded as a precirrhotic state. In the group of malnourished infants, however, periportal fibrosis of an active type associated with cellular infiltration was the characteristic lesion, and present in most cases. In the patients with hepatomegaly and ascites or oedema the severest forms of fibrosis were most frequently found.

Having obtained a rough indication of the type and incidence of hepatic fibrosis in the community, the authors, in an attempt to throw light on the aetiology, directed their attention to the two outstanding environmental factors—namely, nutrition and parasitic infestation. What they reveal is both grim and startling. Owing to the inadequacy of the local

[1] György, P., and Goldblatt, H., *J. exp. Med.*, 1939, **70**, 185.
[2] Himsworth, H. P., and Glynn, L. E., *Clin. Sci.*, 1944, **5**, 93.
[3] Trowell, H. C., and Muwazi, E. M. K., *Arch. Dis. Childh.*, 1945, **20**, 110.
[4] Waterlow, J. C., *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, 1948, No. 263, H.M.S.O.
[5] Walters, J. H., and Waterlow, J. C., ibid., 1954, No. 285, H.M.S.O.
[6] McGregor, I. A., and Smith, D. A., *Trans. roy. Soc. Trop. Med. Hyg.*, 1952, **46**, 403.
[7] Gillman, J., *et al.*, *S. Afr. J. med. Sci.*, 1949, **14**, 21.
[8] —— and Gillman, T., *Perspectives in Human Malnutrition*, 1951, New York.

agriculture, food is never plentiful, and the daily food consumption per head of the population varies from 1,980 calories shortly after the rice harvest to 1,570 shortly before. Protein consumption, which does not exceed 50 g. per day at the best time of the year, is almost entirely of vegetable origin; cows' milk is scarcely consumed at all and fishing is despised. It is not surprising that evidence of severe malnutrition is present at all ages and especially between 6 months and 2 years, when the infant is gradually weaned from the breast and, not yet partaking of the communal food, receives supplementary feeds of sweetened vegetable paps. In addition to this "marginal state of nutrition interrupted by an annually recurring period of privation" the population is subject to an extraordinarily intense parasite infestation, malaria being outstanding. In the survey carried out by I. A. McGregor and D. A. Smith[6] parasitaemia was found to rise from 62% in the first year of life to a peak of 97% from the third to the fifth years and then to fall steadily to about 20% in adult life. Microfilaria, trypanosomiasis, and schistosomiasis are all common, and "helminthic infestations approach malaria in frequency." Infective hepatitis is rare in the Gambia and only four cases were found in three years.

Walters and Waterlow have attempted to identify the various causes of the high incidence of fibrosis, and it is hardly surprising that they cannot incriminate one in preference to another. From their histological and chemical analyses they are apparently justified in excluding prolonged and severe fatty infiltration as a major factor. On the other hand, the consensus of informed opinion is against regarding malaria as a cause of diffuse fibrosis of the kind and severity commonly found in this investigation. The authors therefore feel compelled to fall back on a hypothesis first postulated by the Gillmans in a somewhat different context. From their work on the induction of cirrhosis by repeated injections of Trypan blue,[7] J. Gillman and T. Gillman[8] suggested that two factors are required, one which causes a proliferation of reticulum cells and another which stimulates these cells to form reticulin and collagen fibres. They regard a depressed level of oxidation as this necessary second stimulus. The sequence of events in the Gambian children is then viewed as follows. Severe and recurrent malarial infestation causes the reticulum-cell response of the hepatic mesenchyme. The further progression to active diffuse fibrosis then follows as the result of the depressed level of oxidation due to the impairment of parenchymal-cell function consequent upon the state of malnutrition. In other words, as a result of chronic malaria the reactivity of the hepatic connective tissue to mild damage of hepatic parenchymal cells is greatly enhanced: a degree of parenchymal damage which is usually without effect on the connective tissue now calls forth a progressive fibrogenetic response.

That diffuse hepatic fibrosis can occur in the malnourished in the absence of severe fatty infiltration must not be adduced as evidence that fatty infiltration does not or cannot induce fibrosis in man. The evidence collected by Waterlow[4] himself in the West Indies shows that it almost certainly can. The importance of this work in the Gambia is that it emphasizes that even lesser degrees of malnutrition, themselves incapable of producing chronic liver damage, can do so in subjects whose mesenchymal reactivity has in some way been enhanced. Is it perhaps possible that the frequency of keloidal scarring in certain communities is an indication of this mesenchymal hyperreactivity and should be taken as a warning of their special nutritional vulnerability?

THE HAEMOPHILIAS

Until recently haemophilia seemed to be a well-defined disease. In the eighteenth century Consbruch reported that he had been called to see a boy who had cut his thumb and bled to death within two days. A brother of this boy and several brothers of his mother's had died similarly. The sex-linked inheritance of this liability to bleed to death was thus recognized early. The bleeding tendency has been found to be due to a coagulation defect, the absence of an antihaemophilic globulin necessary for the first stage of coagulation. Normal plasma, which contains this antihaemophilic globulin, restores coagulation when either added *in vitro* or transfused *in vivo*.

The coagulation of the blood has been conveniently divided into three stages, for the first of which the antihaemophilic globulin is necessary: (1) platelets are ruptured and active thromboplastin is formed; (2) active thromboplastin produces conversion of prothrombin into thrombin; (3) thrombin causes the soluble fibrinogen to form elongated crystal-like protein fibrils, which combine into fibres by lateral association. Calcium has long been known to be necessary for the formation of thrombin, and thus there were, besides the antihaemophilic globulin, factors I–IV: fibrinogen, prothrombin, thromboplastin, and calcium. In due course patients were found whose plasma contained all these four factors but who still lacked other agents necessary for coagulation and present in normal plasma. Addition of haemophilic blood could restore coagulation; thus the missing component could not be the antihaemophilic globulin.

[1] *British Medical Journal*, 1952, **2**, 1378.
[2] *Proc. Soc. exp. Biol. (N.Y.)*, 1952, **79**, 692.
[3] Aggeler, P. M., *et al.*, *Science*, 1954, **119**, 806.
[4] Cramer, R., *et al.*, *Acta haemat. (Basel)*, 1953, **10**, 65.
[5] Brinkhous, K. M., and Graham, J. B., *Blood*, 1954, **9**, 254.
[6] Ibid., 1954, **9**, 246.
[7] Rosenthal, R. L., *et al.*, *Proc. Soc. exp. Biol. (N.Y.)*, 1953, **82**, 171.
[8] *J. Lab. clin. Med.*, 1954, **43**, 860.
[9] *Amer. J. Med.*, 1954, **17**, 57.
[10] *Blood*, 1954, **9**, 258.

By comparing these patients' plasma with one another it became clear that there were at least two deficiencies, that of factor V (or V–VI), a labile protein which disappears on storage—hence the synonym labile factor—and of factor VII. Both are hereditary deficiencies, though they are not transmitted by the X chromosome. They are seen rarely, but the clinical importance of factor VII is that its activity is the first to be suppressed during treatment with dicoumarol and ethyl biscoumacetate ("tromexan"). In the laboratory test, deficiency of factors V or VII differs from the classical haemophilia by causing a prolonged prothrombin time. The prothrombin time is also prolonged in hypoprothrombinaemia, usually due to liver disease or vitamin-K deficiency, more rarely congenital. However, when factor V or factor VII is deficient, addition of normal plasma from which prothrombin has been removed can restore the prothrombin time to normal; it will not do so if the plasma under investigation lacks prothrombin.

Among the various terms that have been attached to factor-V and factor-VII deficiency are parahaemophilia A and B, while the term haemophilia itself was reserved for the classical bleeding disorder transmitted with the X chromosome and reversed by transfusion of the antihaemophilic globulin present in normal plasma but not in normal serum. New difficulties in nomenclature arose when it was found that there were differences among the supposedly true haemophilia patients themselves. Plasma from some haemophilics could counteract the clotting defect in others, and this effect was mutual. Thus, although the disease was clinically the same and was inherited with the X chromosome in these groups of patients, it must have been caused by different factors absent in one and present in the other. Whereas the classic antihaemophilic factor was a globulin contained in the fibrinogen fraction of the plasma, the second principle was found both in plasma and in serum. It was described in Britain by Biggs and others,[1] who named it Christmas factor, after the first patient in whom they diagnosed the new condition, and in the U.S.A. by Aggeler and his colleagues,[2] who called it plasma thromboplastin component (P.T.C.). The distinction between classical haemophilia and the new coagulation defect is of practical importance. While both will respond equally to treatment with normal plasma, the second will not be reversed by the therapeutic administration of purified antihaemophilic globulin. Even if normal plasma is transfused an important difference is observed. The classical antihaemophilic globulin disappears within a few days after a transfusion of 500 ml. of normal plasma, but the stability of the beta-globulin, which corrects the new condition, is such that a favourable influence on blood clotting can be observed as long as three weeks after the transfusion.[3]

P.T.C. deficiency and Christmas disease are almost certainly identical disorders, but few authors have as yet stated this with certainty.[4] Some American workers have suggested that the classical haemophilia should be called haemophilia I (or A) and P.T.C. deficiency haemophilia II (or B). Others,[5] who have named factor-V and factor-VII deficiencies haemophilioid states A and B, want to retain for the classical condition the term haemophilia and to call the newly discovered P.T.C. deficiency haemophilioid state C. Aggeler and his group[6] have now proposed the terms haemophilia and deuterohaemophilia, the missing coagulation factors to be called, instead of antihaemophilic globulin and P.T.C., plasma thromboplastin factors A and B (P.T.F.-A and P.T.F.-B). A third plasma thromboplastin factor has been described and was named plasma thromboplastin antecedent (P.T.A.).[7] The new deficiency seems to be distinct from both classical haemophilia and Christmas disease. Its mode of inheritance has not yet been established, but it may not be a sex-linked disorder.

Some might argue that the term haemophilia should comprise all those disorders of coagulation in which there is a defective thromboplastin formation with a normal prothrombin time (to insist on sex linkage might exclude P.T.A. deficiency). It may after all only be a chance that the antihaemophilic globulin was discovered before the beta-globulin, which the American authors have named first P.T.C. and now P.T.F.-B, and which is almost certainly identical with the Christmas factor. Frick[8] restudied fifty-five patients previously simply diagnosed as haemophilics and found that six were in fact cases of P.T.C. and four of P.T.A. deficiency. A similar distribution of the three deficiency states has been found in a survey of twenty-seven families carried out by Rosenthal.[9] On the other hand, Macfarlane[10] is of the opinion that variants of the term haemophilia are confusing and difficult to remember, that it would be best to avoid them, and when no established name like haemophilia is available to refer to deficiencies in terms of the missing factor—"As to the nomenclature of these factors some agreement will have to be reached, and soon, if inextricable confusion is to be avoided."

ADOPTION

Those whose responsibility it is to ensure that the adopted child is given the rights and security of a child in a normal family will find their work greatly assisted by many of the recommendations of the Hurst Committee on adoption[1]. The welfare of the child rather than the satisfaction of the adopters has been the aim of the committee, which has wisely refrained from making sweeping changes which, if interpreted into law, might set up a rigid code that would defeat its own end and increase the number of *de fdcto* adoptions. The committee recommends that the period of notice of placing should be increased from seven to fourteen days and also that the first six weeks of life should not be counted as part of the statutory probationary period. This, together with certain changes of procedure, would make it impossible for an adoption order to be granted before a child is 5 months old. Not everyone has agreed with the committee's insistence that a child should never be able to remember a time when he did not know the truth about his adoption, though psychological disturbance is not an unusual result of sudden or perhaps even harsh revelation to an adolescent that he or she is an adopted

child. The Standing Conference of Societies Registered for Adoption has just issued a useful leaflet for the guidance of adoptive parents entitled, "What Shall We Tell Our Adopted Child?", but if the advice given there is followed—namely, to let the child know "from the beginning" that he has been adopted—there still remains doubt, as Dr. W. J. Pinoff[2] has remarked, about a young child's capacity to understand the implications of the statement (however wrapped up it may be), "I am not your real mother." Dr. F. Charlotte Naish,[3] who is well qualified to speak with authority on the subject, suggests that it would be as well to keep an open mind on this difficult question, while encouraging further research into the best method of telling children that they are adopted.

There is an interesting shift of emphasis in the committee's report from the health of the child to that of the adopters. The adoption of handicapped children need not be discouraged, but, on the other hand, a child should not be adopted by applicants whose good health and expectation of life are in doubt. The suggested compulsory medical examination of the prospective adopters by a doctor appointed by the court is an entirely new departure—which has the approval of the British Medical Association—but its value will be lessened if *x*-ray examination of the lungs is not made obligatory. The committee's desire to ensure that an adoption order is not refused for a handicapped child if the applicants have been informed and understand the implications is commendable. But unfortunately no machinery has been envisaged for simple explanation by the court doctor to the prospective adopters of any salient medical data relating to the child, and it may well be asked how the committee's view that "it is for the applicants to decide when they know the facts" is to be realized unless such facts are explained to them by a doctor.

The most controversial aspect of the report concerns third-party adoption. There has been a general dislike of this method, and the doctor acting as third party has not escaped criticism, in that his interests are often centred more on the psychological needs of the applicants or the welfare of the child's mother than on the best interests of the child himself. This has been given peculiar prominence by a quotation in the report from an unfortunately worded doctor's letter, which, although stated not to be typical of the attitude of the profession generally, has been widely publicized in the press. As the British Medical Association pointed out in the evidence submitted to the committee, the good results achieved in many cases by means of third-party adoption, as well as the impracticability of entirely abolishing direct or third-party placings, call for a realistic approach, and for the provision of safeguards to protect the child in all circumstances. The Hurst Committee's report has been framed on the basic assumption that the welfare of the child must come first. It is to be hoped that this spirit will be retained if the recommendations are embodied in formal legislation.

[1] Report of the Departmental Committee on the Adoption of Children, Cmd. 9248. 1954. H.M.S.O., London. Price 3s. net.
[2] *Lancet*, 1954, **2**, 1285.
[3] Ibid., 1954, **2**, 1229.

MUSCLE SPASM AND RHEUMATISM

It has sometimes been said that, since muscle spasm is partly responsible for the pain of many rheumatic conditions, antispasmodics should be administered. Physostigmine,[1 2] curare,[3] and, more recently, mephenesin,[4 5 6] have all been suggested as suitable, but opinion remains divided on their value, if any, and it is perhaps worth while considering whether there is any sound theoretical basis for their use in rheumatism. It is a well-established clinical observation that in painful conditions of the spine or limb girdles there are local areas of muscle tenderness, often referred to as trigger areas, fibrositic nodules, or myalgic spots. The nature of these is sometimes a matter of diverting speculation, and the fact that local procaine injections are often followed by prolonged or even permanent relief has led to the development of the superficially attractive hypothesis that these are localized areas of muscle spasm which are of themselves responsible for the pain by setting up a vicious circle via a reflex path through the anterior horn cells. This view received some experimental support when Elliott and Kremer[7 8] inserted needle electrodes into the muscles of patients with proved disk lesions and found evidence of spontaneous motor activity restricted to these points. While there was nothing particularly new in the recognition that muscle contraction is associated with referred pain, the emphasis had hitherto been largely on the hyperaesthesia and muscle spasm accompanying visceral lesions. This was the so-called viscero-motor reflex studied by Mackenzie so long ago as 1892.[9] Later Lewis and Kellgren[10 11] in animals and Kellgren[12] in human volunteers observed the presence of muscle spasm and tenderness in response to painful lesions in deep somatic structures, and they concluded that this mechanism probably did not differ fundamentally from that of visceral pain mechanisms. To ascribe to muscle spasm an important part in the production of painful conditions, or at least in their perpetuation, is not, however, quite the same thing. Thus Wolff[13] believed that some forms of headache were due to such a phenomenon, and Schlesinger and Stinchfield[14] suggested that in some cases of acute lumbar disk lesion a vicious circle of pain and muscle spasm might produce a state of affairs which was something more than simple protective splinting. It has been only too easy to suggest that psychological tension leads to muscle tension, giving rise to so-called psychogenic pain. Reviewing these ideas recently Taverner[15] has pointed out that, while an irritative lesion of a nerve root will give rise to spontaneous muscle activity, this is not necessarily associated with pain, and may be observed by the electromyograph in

[1] Cohen, A., Trommer, P., and Goldman, J., *J. Amer. med. Ass.*, 1946, **130**, 265.
[2] Wramner, T., *Acta med. scand.*, 1946, **126**, 241.
[3] Schlesinger, E. B., and Regan, C., *Amer. J. Med.*, 1946, **1**, 621.
[4] Hermann, I. F., and Smith, R. T., *Lancet*, 1951, **71**, 271.
[5] Berger, F. M., and Schwartz, R. P., *J. Amer. med. Ass.*, 1948, **137**, 772.
[6] Schlesinger, E. B., *et al.*, *Trans. Amer. neurol. Ass.*, 1948, **73**, 94.
[7] *Lancet*, 1944, **1**, 47.
[8] Ibid., 1945, **1**, 4.
[9] *Med. Chron.*, 1892, **16**, 293.
[10] *Clin. Sci.*, 1939, **4**, 47.
[11] Ibid., 1939, **4**, 35.
[12] Ibid., 1938, **3**, 175.
[13] *Harvey Lect.*, 1943–4, **39**, 39.
[14] *J. Bone Jt Surg.*, 1951, **33A**, 480.
[15] *Ann. rheum. Dis.*, 1954, **13**, 331.
[16] *Acta chir. scand.*, 1951 .160.

pain-free muscles. By contrast, in the patient with acute lumbago at rest the muscles may be completely silent. The observations of Frykholm[16] on brachial neuralgia due to nerve root compression in the cervical region are of the greatest interest and throw some new light on this problem. He was able to demonstrate at operation in this type of case that stimulation of the ventral root gives rise to deep pain associated with muscle tenderness of the so-called myalgic type, while stimulation of the dorsal root gives rise to a more peripheral pain associated with paraesthesiae—that is to say, pain of a neuritic character. Perhaps muscle spasm therefore, at least in rheumatic conditions, is no more than a result of referred pain or of ventral root irritation.

OSTEOPOROSIS AND CORTISONE

Osteoporosis—a condition caused by loss of the proteinous matrix of bone—is a common finding in Cushing's syndrome, and F. Albright[1] has produced evidence that this is due to an excess of certain adrenocortical hormones, such as hydrocortisone, which have an anti-anabolic action whereby they prevent the building up of proteins from amino-acids. In sufficient dosage over a prolonged period of time cortisone or corticotrophin induces many of the changes found in Cushing's syndrome, and workers at the Mayo Clinic[2] have shown that in some patients a dose of cortisone exceeding 100 mg. daily causes a negative nitrogen and calcium balance. In patients with rheumatoid arthritis this undesirable side-effect of cortisone is often counteracted by an increase of appetite, which leads to enhanced protein intake and hence a positive rather than a negative protein balance. Nevertheless it was not unexpected that in some patients prolonged administration of cortisone should lead to osteoporosis, with pain in the back and collapse of vertebral bodies, and more than a dozen such cases have now been reported.[3–7] In many instances it is difficult to be certain that the condition is attributable solely to the cortisone, because osteoporosis may occur as a complication of rheumatoid arthritis, during prolonged immobilization, and particularly in postmenopausal women. In some patients the symptoms of osteoporosis and pathological fractures have developed within a few months of the start of cortisone treatment, and possibly the increased sense of well-being produced by the drug has led patients with osteoporosis from other causes to indulge in unaccustomed activity which before treatment was prevented by pain and muscle spasm. Recently osteoporosis and crush fractures of the vertebrae have been reported[7] in four male patients receiving prolonged cortisone or corticotrophin—one of them being a boy aged 9—and there can be little doubt that the treatment caused the bone changes. This hazard should be kept in mind when cortisone treatment is prolonged and particularly when large doses are given to elderly patients or postmenopausal women; physiotherapy[8] and testosterone[2] may help to avert it.

[1] *Harvey Lecture Series*, 1943, **38**, 123.
[2] Sprague, R. G., *et al.*, *Arch. intern. Med.*, 1950, **85**, 199.
[3] Demartini, F., *et al.*, *J. Amer. med. Ass.*, 1952, **149**, 750.
[4] Teicher, R., and Nelson, C. T., *J. invest. Derm.*, 1952, **19**, 205.
[5] Soffer, L. J., and Bader, R., *J. Amer. med. Ass.*, 1952, **149**, 1002.
[6] de Sèze, S., *et al.*, *Presse méd.*, 1953, **61**, 819.
[7] Curtiss, P. H., *et al.*, *J. Amer. med. Ass.*, 1954, **156**, 467.
[8] Clark, W. S., *et al.*, *J. clin. Invest.*, 1954, **33**, 505.

TETANUS IMMUNIZATION

Every year since the war about seventy people in England and Wales have died of tetanus, and probably at least as many more have recovered from the infection. The highest death rate was in boys aged 5–14 years, and most of the deaths were of people living in rural areas, particularly in East Anglia and the south-west. Some of these deaths might have been prevented by more extensive use of prophylactic antitoxin, but the experience of Cole[1] suggests that many probably had injuries so trivial that they were not even seen by a doctor. Every year many thousand injections of tetanus antitoxin are given and are followed inevitably by a number of serum reactions; occasionally one of them is fatal. Every day doctors are faced with the difficult task of weighing the risks of horse serum against those of tetanus.

There is strong though indirect evidence for the value of active immunization against tetanus. Only twelve cases occurred in the American Army during the last war, and only six of these had received the basic course of toxoid.[2] The American Army used no antitoxin, but relied entirely upon booster doses of toxoid after injury. Further suggestive evidence was obtained by Ipsen,[3] who showed that there has been a marked decline in tetanus morbidity in Massachusetts in children under 5 years of age, most of whom are now actively immunized, and in young adults, a high proportion of whom were actively immunized in the armed Forces. Conybeare and Logan[4] found a similar decline since the war in the tetanus death rate of young male adults in England and Wales.

Active immunization on a scale sufficient to produce an appreciable result would hardly be practicable were it not for the development of vaccines combining tetanus toxoid with other antigens already widely used. The immunization of all infants with a single vaccine effective against diphtheria, whooping-cough, and tetanus, with later boosting doses as required, would be ideal, but it is important that in combining antigens nothing should be lost. Barr and Llewellyn-Jones showed[5] that the result obtained with a combined prophylactic depends to an extent not fully understood on the composition of the vaccine and the state of immunity of the inoculated. However, many studies have shown that it has been possible to produce satisfactory levels of antibody against the component parts of combined vaccines, so the problem should not be insuperable. Field trials are in progress at present to confirm that the protective effect of a whooping-cough vaccine is not diminished by combining it with a diphtheria antigen. A triple vaccine incorporating tetanus toxoid would require similar trial.

We record with regret the death on January 25 of Dr. Charles Herbert Hampshire, C.M.G., secretary of the British Pharmacopoeia Commission from 1929 to 1950.

[1] *Practitioner*, 1951, **167**, 247.
[2] Long, A. P., *Amer. J. publ. Hlth*, 1948, **38**, 485.
[3] *New Engl. J. Med.*, 1954, **250**, 37.
[4] *British Medical Journal*, 1951, **1**, 504.
[5] *Brit. J. exp. Path.*, 1953, **34**, 12.

EMERGENCIES IN GENERAL PRACTICE

THE MANAGEMENT OF ECLAMPSIA

BY

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Although eclampsia remains one of the most serious of obstetrical complications, the Australian workers have shown that in the practice of a first-class maternity hospital a determined effort to eliminate eclampsia may be completely successful.* The measures which succeeded at the Crown Street Hospital in Sydney were administrative as well as medical. They involved arrangements for antenatal out-patient care at the very highest standard, and the reallocation of beds so as to increase antenatal accommodation at the expense of lying-in beds. The latter allowed the admission of all patients who showed evidence of pre-eclamptic toxaemia, and also many of those patients who were believed to be likely to develop pre-eclampsia, on the basis of excessive weight gain observed at their frequent antenatal examinations. The most important obstacle to the application of such methods in this country occurs in areas where maternity hospital beds are insufficient to allow an adequate number to be set aside for antenatal patients. In hospitals in more favoured areas eclampsia is already rare, while a general practitioner carrying out alert antenatal care very seldom needs to treat an eclamptic patient at home. Although successful measures for the complete prevention of pre-eclamptic toxaemia have not yet been established, the prompt treatment of early cases of toxaemia is often successful and the immediate transfer to hospital of patients showing severe pre-eclampsia is enough in itself to make convulsive eclampsia a very great rarity in domiciliary practice.

Space does not permit of elaboration of the conservative treatment of pre-eclampsia. So far as general practice is concerned the most successful measure is to increase the time spent by the patient in bed to the maximum compatible with her domestic circumstances. Restriction of sodium intake in the form of cooking salt, table salt, and sodium-containing digestive medicaments is helpful but not always easy. A nocturnal barbiturate —for example, butobarbitone 0.18 g. (3 gr.)—is often advisable. It is most important to keep very clearly in mind the indications that a case of pre-eclampsia is becoming too severe to handle in the patient's home. Of these the most important are (*a*) oedema which persists in the lower limbs after a complete night's rest, or oedema which significantly affects the hands or face; (*b*) a blood pressure at or above the level of 150/100; (*c*) albuminuria confirmed in a catheter specimen of urine, no matter how small the quantity of albumin may be; and (*d*) finally the well-known prodromal symptoms of eclampsia itself, splitting headache, blurring of vision, severe epigastric pain, and reduction in output of urine.

General Principles of Management

The general practitioner will wish to undertake domiciliary treatment of eclampsia only in most isolated communities, the number of which has been greatly reduced by the availability of good ambulance services. According to Frew the eclamptic patient appears to travel well by air if under the action of a basal narcotic such as bromethol.

There is as yet no specific treatment for eclampsia. It is universally accepted that measures to limit the number of the fits offer the best hope of saving life. Along with these measures attention is required to the prevention or prompt treatment of such complications of eclampsia as inhalational pneumonia, cardiac embarrassment, or suppression of urine.

The sedative regime associated with the name of Stroganoff has been widely employed with varying success. Morphine and chloral were the drugs originally recommended, but other and more powerful narcotics have been introduced without modifying the essential principles. Of these later drugs the most valuable appear to be sodium thiopentone and bromethol. All are potential respiratory depressants, and the balance between the effective and the toxic dose is very delicately poised.

Certain drugs which reduce the elevated blood pressure are also effective in preventing convulsions. Of these, the extract of veratrum viride (a plant of the lily family) manufactured under the proprietary name of "veratrone" has always been popular in the Cincinnati school. In this country Stern and Burnett have authoritatively recommended it.†

Whichever type of drug is selected, the general nursing management of the patient is of the greatest importance. The maintenance of a clear airway is essential. Artificial dentures should be removed as soon as possible. The mouth must be kept clear of secretion, blood, or vomitus. The use of the semiprone position, changing from one side to the other at two-hourly intervals, is probably sound, but care must be taken to swab out the dependent cheek before moving the patient. Quiet, the avoidance of bright light, and the maintenance of an even temperature help to diminish the afferent stimuli which may precipitate convulsions. The eclamptic patient should not be overheated, and there is no reason to encourage sweating by the application of a harsh blanket. The vigorous perspiration often seen during recovery from eclampsia is the result of that recovery, and not its cause.

The possibility of inhalational pneumonia is diminished by good nursing. It is important to give neither solids nor liquids by mouth during the acute phase in order to prevent their inhalation during feeding or regurgitation. Penicillin may be given prophylactically in dosage of 300,000 units eight-hourly.

The heart is most effectively supported by ensuring that anoxia is reduced by keeping the airway completely unobstructed in the intervals between fits. If available, oxygen should be administered immediately after each convulsion and continued so long as there is the slightest trace of cyanosis. Digoxin 1.5 mg. (1/40 gr.) may be given intravenously to a patient who has not been receiving any other form of digitalis. Venesection of 500 ml. may be useful in acute pulmonary oedema, which occasionally complicates cardiac failure in eclampsia.

Obstetrical intervention is generally restricted to the terminal second stage. There is no doubt that the patient who has an eclamptic convulsion when the head is on the perineum is often treated most effectively by an immediate forceps delivery. In other circumstances the better interests

*Hamlin, R. H. T., *Lancet*, 1952, **1**, 64.

†*J. Obstet. Gynaec. Brit. Emp.*, 1954, **61**, 590.

of the patient are usually served by conservatism. In hospital practice there is a place for induction of labour in antepartum eclampsia, but usually such intervention is delayed until the fits are controlled by sedatives, by which time labour has often ensued spontaneously. Caesarean section is not to be regarded as a treatment of choice in any circumstances. It is inevitably associated with a high maternal mortality in eclampsia. There are occasional cases, however, where it may seem justified to a person with very great experience of the treatment of eclampsia.

Observation of the eclamptic patient must be close and uninterrupted. Changes in skin colour, the depth of coma or degree of restlessness, the temperature, pulse and respiration, the variations in blood pressure, the volume of urinary secretion, the activity of the uterus, all require watching. Too much disturbance of the patient is to be avoided, but a four-hourly report from the midwife is a great support in controlling treatment. There is little point in being scrupulous about the foetal heart sounds during the acute phase of the disease, for the foetus is always in grave danger in eclampsia and the detection of signs of foetal distress is unlikely to influence treatment greatly. Despite the old adage, intrauterine death of the foetus does not have much influence on the maternal prognosis.

First Aid

The fit of eclampsia is essentially similar to that of epilepsy except that there is no phenomenon strictly comparable to the aura of epilepsy. After slight facial twitching a tonic phase sets in, being followed by the onset of clonic spasms. The end of the fit is usually preceded by the coincidence of a clonic inspiratory effort with relaxation of the muscles controlling the glottis. Cyanosis at once diminishes. A moment or two later the jaw muscles relax completely and the fit may be said to have passed off. Coma of variable length and depth follows. In comparison with post-epileptic coma it is relatively brief.

The primary concern of first aid is the prevention of injury during the clonic phase. Gentle restraint for the writhing body will prevent the patient rolling out of bed or knocking her limbs against walls or furniture. Little more than this is possible. The advice to force a gag, a wedge of wood, or the handle of a spoon between the teeth is sound enough in theory, but usually the clenching of the jaws is much too firm to allow this to be done without the risk of loosening teeth or causing other damage. It is some comfort to recollect that if the tongue lies between the teeth at the moment of onset of the fit, nothing can save it, while if it is internal to the opposed jaws it is in any case safe. The time for insertion of a gag or dental prop is immediately after the fit, opportunity being taken to remove any artificial dentures and to swab out the mouth and pharynx if the tongue has been bitten or if there has been any regurgitation of gastric content.

The intravenous administration of sodium thiopentone in dosage of 0.25 to 0.5 g. is very sound as a first-aid procedure. It may be expected to arrest the fit at any stage, and by its prompt administration some lives may be saved which would otherwise be lost during a single unusually severe eclamptic convulsion. Unfortunately the preparation of the injection takes a little time, so that a first convulsion may have come and gone before it can be administered. If this is the case it is prudent to put down the syringe containing the injection in some safe but accessible position, so that it will be instantly available if there be a further attack.

At one time it was customary to endeavour to control the fits by the exhibition of prodigious quantities of chloroform. There is little doubt that the liver damage of eclampsia was often fatally aggravated by this additional insult. Inhalational anaesthetics are useless during the respiratory arrest of the convulsion. Their employment immediately after the convulsion is justifiable as a first-aid measure to delay the onset of further attacks where fits are following each other in very rapid succession and thiopentone is for any reason unavailable. Chloroform on a Schimmelbusch mask at the rate of approximately 15 drops per minute for not more than 2–3 minutes may allow time for less damaging sedatives to take effect. Ether, theoretically preferable, is too apt to produce salivation, an added embarrassment to breathing.

The immediate management of the eclamptic patient includes certain examinations. It is most useful to obtain some idea (*a*) whether the patient is in labour or likely soon to be in labour, (*b*) whether there is any abnormality likely to give rise to mechanical difficulty, and (*c*) whether her urinary output is satisfactory. As the necessary examinations may well precipitate a convulsion in a restless semi-conscious patient, it is well to take the opportunity to carry them out in the period of coma immediately following a seizure, especially if thiopentone or chloroform has been given. The unconsciousness is then likely to be prolonged by a few additional minutes which will allow a quick abdominal and vaginal examination and the passage of a catheter. If a self-retaining catheter is available, its use should certainly be considered. A clip can be applied to it when the bladder has been emptied, and thereafter the release of the clip at four-hourly intervals enables the urinary output to be exactly measured without further disturbance of the patient.

Further Management

When "Flying-Squad" Service Available

If such a service operates in the practitioner's area and is willing to undertake treatment of the eclamptic prior to and during her movement to hospital, then the hospital should be contacted without delay. A joint scheme of treatment can be worked out in accordance with local policy.

No Flying-Squad Available but Removal to Hospital Possible

In city practice it is enough to move the patient as quickly as possible by ambulance from her home to the hospital of choice. The practitioner should accompany his patient. It is useful to take a gag and tongue forceps, a Schimmelbusch mask, and a bottle of chloroform. Where distances are greater it is wiser to begin sedative treatment in the home and to delay moving the patient until such sedatives have had time to take effect. A very useful drug for this purpose is bromethol, of which 5.5 ml. dissolved in 220 ml. of tap-water at body temperature may be administered rectally. This acts within 20 minutes and the deep narcosis which follows renders the transport of the patient fairly safe. Unfortunately, the use of bromethol in general practice is limited by the fact that it is now rarely obtainable outside maternity hospitals, its vogue as a basal anaesthetic in general surgery having expired. It is an excellent "flying-squad" medicament, but the unaided general practitioner will often need to adopt other expedients. Of these morphine with all its faults as a respiratory depressant is the most useful and generally available. Its reinforcement with some other sedative (e.g., chloral or a barbiturate) is usually practised, since it may then be possible to avoid very full doses of morphine. The following scheme of dosage is advised, the hour of commencing treatment being represented by the symbol "H." All drugs are administered intramuscularly.

Hour	Drug	Dose
H	Morphine	15 mg. (¼ gr.)
	Sodium phenobarbitone	0.3 g. (5 gr.)
H+1	Morphine	10 mg. (1/6 gr.)
H+2	*Depart by ambulance*	

The second dose of morphine should be omitted in the unlikely event of any real respiratory depression (e.g., respirations 10 per minute or less), but if so an appropriately loaded syringe should be entrusted to the ambulance nurse to use if the patient has a further seizure during transit. It must be emphasized that the delay in moving the patient is justified only if the first injection produces some degree of control of her fits. If she continues to have fits some thirty minutes after starting treatment, no time should be lost in giving the second injection of morphine and dispatching her to hospital.

Removal to Hospital Impossible

Faced with the need to treat his patient entirely at home, the single-handed practitioner cannot hope to apply treatments which may tax the resources of a fully-staffed maternity hospital. Methods involving prolonged medical supervision can hardly be selected, and it is not practical to consider such treatments as the continuous intravenous thiopentone management of O'Donel Browne. The drugs readily available to the practitioner are likely to be limited to those of sedative type, and accordingly a Stroganoff type of management is likely to be forced on him. The aim should be to keep the patient deeply narcotized for at least 24 hours, and thereafter to prolong the sedation in lesser degree for a further 24 hours. Most patients are out of danger by that time. The following dosage scheme is suggested, the hour of commencing treatment being represented by the symbol "H"; all drugs are administered intramuscularly.

Hour	Drug	Dose
H	Morphine	15 mg. (¼ gr.)
	Sodium phenobarbitone	0.3 g. (5 gr.)
H+1	Morphine	10 mg. (1/6 gr.)
H+4	Morphine	15 mg. (¼ gr.)
H+8	Morphine	15 mg. (¼ gr.)
H+12	Sodium phenobarbitone	0.3 g. (5 gr.)
H+18	Morphine	15 mg. (¼ gr.)
H+24	Sodium phenobarbitone	0.3 g. (5 gr.)
H+30	Morphine	15 mg. (¼ gr.)
H+36	Sodium phenobarbitone	0.3 g. (5 gr.)

The recurrence of fits despite treatment might well justify the acceleration or increase of the doses of morphine. Indications to delay the next dose include undue respiratory depression (respirations 10 or fewer per minute), persistent cyanosis, and accumulation of bubbling secretion in the trachea and bronchi—usually an indication for full dosage of atropine (i.e., 1 mg.—1/60 gr.).

In addition to sedatives, some value is attached to the parenteral injection of hypertonic solutions of glucose or magnesium sulphate. These may have some effect in reducing cerebral oedema. Glucose is believed also to favour diuresis, while magnesium sulphate may reduce blood pressure slightly and appears to potentiate the anticonvulsant action of morphine. Glucose is given intravenously in daily dosage of 1,000 ml. of 20% aqueous solution. Magnesium sulphate is usually given intramuscularly in 50% aqueous solution, in dosage of 10 ml. repeated after each convulsion up to a total daily dose of 60 ml. The side-effect of paralysis of voluntary muscle must be kept in mind, and a total dose of 20 ml. should not be exceeded without making sure that the knee-jerks are present at the time each fresh injection is given.

Other useful drugs are: (*a*) *Sedatives:* Chloral hydrate, oral or rectal, 3 g. (45 gr.)–4 g. (60 gr.); paraldehyde, rectal, 40 ml. (1 fl. oz.), intramuscularly, 10–20 ml. (2–4 fl. dr.). (*b*) *Veratrone,* intramuscularly, 0.6 ml. (10 minims)—as initial dose. Further doses, if necessary, may be started after 30 minutes. They must be controlled by blood-pressure readings at 15-minute intervals. The aim is to secure an early reduction of systolic blood pressure to less than 120. Thereafter veratrone is withheld unless the systolic pressure rises above 150 mm. Hg. For details see Stern and Burnett's paper mentioned earlier in this article.

Next article on Emergencies in General Practice.—"Acute Lumbago and Sciatica," by Mr. John Charnley.

Refresher Course Books.—Copies of the first two volumes of collected articles from the Refresher Course for General Practitioners published in the *Journal* are still available at 25s. (postage 1s.) each. The first volume contains 55 articles and the second 60. Each article has been revised and brought up to date by its author.

These books may be obtained from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1, or through any bookseller.

INTERNATIONAL FEDERATION OF GYNAECOLOGY AND OBSTETRICS

The constitution of the International Federation of Gynaecology and Obstetrics has now been adopted by 42 countries, including Great Britain, Canada, Eire, India, New Zealand, the United States, and Russia. The Federation was founded during the International Congress on Obstetrics and Gynaecology in Geneva last year, and has the following objects:

(*a*) To promote the development of science and assist in scientific research work relating to all the fields pertaining to gynaecology and obstetrics, to further the attainment, by all appropriate means, of a higher level of physical and mental health of women, mothers and their children, to develop and improve the exchange of information and ideas in the field of gynaecology and obstetrics, to contribute to the research of fresh knowledge in this field, to contribute to the improvement of teaching standards in the profession, to promote international co-operation and facilitate relationships between national medical bodies of the profession.

(*b*) To recommend a common policy in regard to these matters on the proposal of member societies.

(*c*) To represent member societies whenever joint scientific action can be pursued in such matters.

(*d*) To foster the initiation and development of all activities aiming at the attainment of the objects of the Federation as defined under (*a*) above.

(*e*) To organize international congresses, including all countries or certain groups of countries. Circumstances permitting, each international congress shall be held successively on a different continent. Members of national societies may attend all congresses.

The executive board of the Federation is composed as follows: *president,* Professor H. de Watteville (Switzerland); *vice-presidents,* Professor L. Gérin-Lajoie (Canada) and Professor E. Cova (Italy); *secretary,* Professor R. Keller (France); *treasurer,* Dr. F. L. Adair (U.S.A.); *members,* Professor César A. Brea (Argentina), Professor O. Rodrigues Lima (Brazil), Professor H. Martius (Germany), Dr. V. B. Green-Armytage (Great Britain), Professor B. N. Purandare (India), Professor Hideo Yagi (Japan), Professor A. Bravo (Mexico), Dr. J. Harbutt (New Zealand), Dr. Howard C. Taylor, jun. (U.S.A.), and Professor A. Belochapko (U.S.S.R.); *executive secretary,* Dr. W. Geisendorf (Switzerland).

ORDER OF ST. JOHN OF JERUSALEM

The *London Gazette* has announced the following promotions in, and appointments to, the Venerable Order of the Hospital of St. John of Jerusalem:

As Knights: Brigadier R. A. Hepple, C.B.E., M.C., M.B., and Dr. W. P. Warner, C.B.E., D.S.C. *As Commanders (Brothers):* Surgeon Captain E. H. Lee, M.D., Brigadier K. A. Hunter, O.B.E., M.D., Lieutenant-Colonels G. N. Morris, M.B., F.R.C.S., and F. K. Mugford, M.B., Air Commodore A. A. G. Corbet, M.D., Mr. K. G. W. Saunders, O.B.E., F.R.C.S., and Drs. S. H. Keshen, A. P. Gorham, E. M. Dearn, R. A. P. Gray, and R. R. Trail, C.B.E., M.C. *As Officers (Brothers):* Colonel G. A. Winfield, O.B.E., M.D., Lieutenant-Colonels S. Mackenzie, O.B.E., M.R.C.S., L.R.C.P., J. A. Melanson, M.B., and W. R. Everatt, T.D., M.B., Major M. J. Bett, M.D., Professor J. Galea, M.B.E., M.D., Messrs. J. S. Grant, M.D., F.R.C.S., J. A. White, M.B., F.R.C.S., V. C. J. Harris, F.R.C.S., A. L. Buchanan, M.B., F.R.C.S., R. H. Percival, F.R.C.S., and G. E. G. Peirce, M.B., F.R.C.S., and Drs. R. S. Reid, M.B.E., R. A. Dench, M.C., J. Morris, F. O'Daly, J. C. Moor, J. C. Cassaglia, G. D. E. Tullis, A. H. Penistan, E. F. Brown, E. L. Butler, M. Y. Paget, G. E. Mullins, R. Dick, R. M. B. MacKenna, H. G. Wiltshire, L. J. Haydon, T.D., H. E. Offord, J. R. Oddie, J. T. Watkins, J. H. Dixon, J. J. Kennedy, V. C. Meyer, J. McC. Caldwell, and H. E. W. Roberton. *As Associate Officers (Brothers):* Drs. H. M. Jacobs, A. Rabinowitz, and E. B. Doany. *As Officer (Sister):* Dr. Eily E. Gurr. *As Serving Brothers:* Surgeon Captain F. G. W. MacHattie, Surgeon Commander M. Wellman, Colonels J. E. Andrew, M.D., F.R.C.S., and E. E. Tieman, O.B.E., M.D.,

Wing Commander H. J. Bright, M.D., Squadron Leader D. O. Coons, M.D., Messrs. J. J. Coulthard, M.B., F.R.C.S., and M. H. Elliot-Smith, M.B., F.R.C.S., and Drs. O. J. Curl, K. O'L. Doherty, W. J. Boyd, T. E. Kirk, H. S. Sharpe, R. C. Wilson, P. de Bec Turtle, D. O. Dickie, J. S. Stuart, H. Leslie, L. D. M. Gavin, J. F. M. Milner, V.D., J. W. Dowzer, H. W. Taylor, R. E. Crockatt, S. C. Pritchard, O.B.E., D. W. R. Richardson, V. St. C. Lucas, O. Thomas, D. T. Clark, J. W. McAllan, R. A. Lewis, A. Sandison, J. R. Martin, and R. W. Cunningham. *As Associate Serving Brothers:* Drs. A. Gordon and Kwan Kui Lun. *As Serving Sister:* Dr. Eleanor M. Whitehead.

Reports of Societies

RECENT ADVANCES IN BRAIN SURGERY

The Section of Psychiatry of the Royal Society of Medicine held a joint meeting with the Section of Neurology on January 11 to hear papers on three aspects of recent research made possible by modern brain surgery. Professor W. MAYER-GROSS, president of the psychiatry section, was in the chair, and was joined on the platform by Professor F. J. NATTRASS, president of the neurology section. The first paper was by Professor ALFRED MEYER, head of the neuropathology department at the Maudsley Hospital, on "Temporal Lobe Epilepsy and the Hippocampal Region."

Local Sclerosis in Temporal Lobe Epilepsy

The hippocampus, or Ammon's horn, was the most medial part of the temporal lobe of the cerebral hemisphere, said Professor Meyer. In 1927 Spielmeyer had shown that interference with its blood supply or anoxia (as in a major epileptic fit, carbon monoxide poisoning, and similar conditions) led to its sclerosis—selective or even total neuronal loss and glial proliferation. The same year had seen the beginning of the overthrow of the traditional theory that the hippocampus is part of the "smell" brain, subserving olfactory function: it was now regarded as part of the "visceral" or limbic brain, concerned with the control of the autonomic functions of the body. In this the hippocampus formed part of a system with the uncus, the adjacent amygdaloid nucleus, the anterior uvula, the anterior cingulate region, and the thalamus and hypothalamus.

Penfield and his collaborators had recently introduced the operation of anterior temporal lobectomy for epileptics who showed no gross focal lesion, continued Professor Meyer. In their series two-thirds of all such cases showed sclerosis of a part of the temporal lobe, where in their view the lobe had at some time, probably at birth, been wedged a little way through the notch or incisura of the tentorium and its blood supply compressed. All cases with "incisural" sclerosis in the removed lobe had clinical or electroencephalographic evidence of temporal lobe epilepsy. Professor Meyer, in collaboration with Dr. Denis Hill, Mr. Murray Falconer, and Mrs. E. Beck, had been making a similar study of the tissue removed at operation for temporal lobe epilepsy. Out of 18 cases of non-focal or "incisural" epilepsy 12 showed the predicted hippocampal sclerosis, but the other 6 had very little specific abnormality in the removed lobes. Of course it was possible that in these last cases the damage lay elsewhere—perhaps in the temporal lobe on the other side, which had not been opened. Study of a comparable group of epileptics with focal lesions of different kinds (four with angiomata, two with oligodendrogliomata, and so on) which produced the well-known uncinate fits showed lesions much more frequently also in the uncus and amygdaloid nucleus and less commonly in the hippocampus. However, a sclerosis of the hippocampus could extend forwards to the uncus and into the amygdaloid nucleus, as had, in fact, been demonstrated by the speaker in four of the few biopsy specimens in which amygdaloid tissue was available. He had also seen in a necropsy on a demented child of 8, who had suffered repeatedly from status epilepticus, sclerosis of the hippocampus (Sommer sector areas H3 to H5) as well as softening of the amygdaloid nucleus, visible to the naked eye. In a woman of 43, who began to have severe epilepsy six months after prefrontal leucotomy for chronic mania and survived for seven years, the amygdaloid nucleus in addition to the uncus and hippocampus was found sclerosed.

It was interesting that Gastaut and Magnus and their associates had found that electrical or chemical stimulation of the amygdaloid nucleus in animals was the best way to elicit a psychomotor (temporal lobe) fit experimentally. Professor Meyer pointed out that this nucleus, the uncus, and the hippocampus received blood through anterior branches of the anterior choroidal artery, whereas posterior branches of this artery supplied the globus pallidus, which in his post-mortem cases was histologically normal. He doubted whether hippocampal sclerosis occurred always at birth, as proposed by Penfield, but thought it might well be the sequel to anoxia from any cause, and particularly from the onset of severe major epileptic fits, whenever these first occurred.

Results of Cingulectomy

Dr. C. W. M. WHITTY, of the Radcliffe Infirmary, Oxford, spoke on "Cingulectomy," the removal of the anterior part of the cingulate gyrus (area 24 of Brodmann), which, as already mentioned by Professor Meyer, formed a part of the limbic brain.

Bilateral cingulectomy in monkeys produced no neurologically detectable changes but considerable emotional alteration: the animals lost their aggressiveness and became very tame. They lost all normal fear reactions, and their social behaviour with their cage-mates was different. It was thought, therefore, that the operation might be useful in the treatment of psychiatric disorders in man, and since 1944 35 human cases had been studied, the earlier operations done by the late Sir Huge Cairns, the more recent by Mr. Walpole Lewin. A limited subpial suction ablation of the cortex was made, about 4 cm. × 1 cm. × 1 cm. of tissue being removed, great care being taken to preserve branches of the anterior cerebral artery. Necropsy of five patients who later died showed that the intended area had been accurately excised in all but one. Le Beau, of Paris, had published his results on 30 cases, but the excised area was bigger.

The Oxford workers first operated on 18 chronic deteriorated mental cases, 11 of whom were schizophrenics. Aggressive outbursts and obsessional symptoms became fewer, but the relief was only temporary. They then treated 17 patients who were in hospital on account of single severe anxieties or compulsive or obsessional symptoms but whose basic personalities were well preserved. Of 7 cases in the anxiety group 4 were improved—that is, they were happier, able to go home and even work—and 2 greatly improved (all symptoms abolished). Of 10 obsessionals 7 were greatly improved and 2 improved. Improvement was not immediate, but took place gradually over a number of months. As the obsessions faded away, emotional reactions became more open and less persistent, and drive and power of concentration lessened. Follow-up after one to four years showed that patients had lost their tendency to abnormal emotional or cognitive perseveration and also some of their inhibitions. Dr. Whitty thought there was less blunting of personality than after frontal leucotomy or orbital undercutting, but he was very cautious in making any claims for cingulectomy. There seemed to be a tendency to bring out latent epilepsy, at least temporarily; though only one case had persistent fits post-operatively. During the operation electrical stimulation of the area to be excised produced variable results—stomach sensations in some, a drop in blood pressure or sweating attacks in others. Study of blood pressures, pulse rates, and response to adrenaline and mecholyl before and after operation suggested that cingulectomy produced a change in the range of response but not a change in the baseline.

Stereotactic Brain Surgery

Professor H. HASSLER, head of the neuropathology department at Freiburg University (Germany), showed a film on "A New Stereotactic Method of Brain Surgery." Different areas in the thalamic nuclei (or elsewhere in the brain) could be accurately coagulated by means of a specially introduced diathermy needle which could also be used to take electrical recordings from the area, or to stimulate it, before producing the coagulation. A rigid metal ring was fixed round the patient's skull in the plane of the auditory meatuses and the root of the nose, and an aiming-bow (a metal half-ring running over the vertex and attached to the base ring) fitted to carry the needle. Air encephalograms of the patient's brain were taken to measure off various dimensions of his brain and compare them with a model brain which could be placed inside a duplicate base-ring and aiming-bow. From the model it was estimated at what angle to set the aiming-bow, and where it should carry the operation needle, and how far this should plunge in. After all this preparation the needle could be simply slid into position through a previously prepared skull defect, and a second air encephalogram would show that it had attained the desired place. This could be confirmed by the effects of electrical stimulation through the needle in the conscious patient. The film showed dramatically the results in one or two chosen cases out of the 140 so far treated, most of them for intractable pain, or for muscular rigidity, tremor, or athetosis. For example, a man with typical Parkinsonian rigidity and intention tremor of a severe degree had the needle introduced into the anterior ventral nucleus of the thalamus first on one side and, three days later, on the other. Electrical stimulation of one nucleus inhibited the rest tremor of limbs on the other side of the body. After coagulation of both nuclei the muscular rigidity was abolished, walking became very much easier, intention tremor almost vanished, but in the right arm tremor at rest persisted, though diminished. A girl with severe athetosis from a birth injury had her spontaneous uncontrollable movements very greatly reduced by coagulation of a segment of the globus pallidus.

Discussion

In the discussion Dr. M. A. PARTRIDGE paid tribute to the extraordinary accuracy of Professor Hassler's technique, and wondered whether cingulectomy would compare favourably with rostral leucotomy. Mrs. A. PETRIE said that patients with rostral leucotomy gave a very different pattern of response to her battery of personality tests from Le Beau's cingulectomy patients whom she had recently tested: the latter resembled patients on chlorpromazine. Dr. J. S. PIPPARD said his study of 240 cases of rostral leucotomy clinically showed only slight personality changes, and these were similar to the changes described by Dr. Whitty. He wondered what Mrs. Petrie's tests were measuring. Dr. STEPHEN SHERWOOD described how they had done 100 cingulotomies at Severalls Hospital, Colchester, the results of which compared favourably with classical leucotomy. He also gave details of their attempts at stereotactic brain surgery.

LOCAL MEDICAL SOCIETIES

Persian Gulf Medical Society

The third annual meeting of the society was held in Bahrain, Persian Gulf, on November 24 and 25, 1954. Over 70 delegates attended, representing governments, oil companies, and the American Mission, from countries extending from Iraq in the north to Muscat in the south of the Gulf.

The Congress was officially opened by the Ruler of Bahrain, SHEIKH SIR SULMAN BIN HAMAD AL-KHALIFAH. The guest speaker was Dr. HOEFNAGELS, of the World Health Organization, who read a paper entitled "The Position of Tuberculosis Control as Part of a General Health Programme." It has become an established policy of the society to lay emphasis on practical problems of the Persian Gulf area, and this was well shown by the choice of papers. Dr. JOYCE OWEN, of the Kuwait Oil Company, followed Dr. Hoefnagels with a paper on "The Out-Patient Treatment of Tuberculosis amongst Arab Women and Children." Dr. JAMES GRANT, of the Bahrain Government, Public Health Department, read a paper on "The Susceptibility of Insects to Insecticides." Dr. WELLS THOMS, of the American Mission, who has spent most of his life in the area, read an interesting paper on his experience in dealing with leprosy in Muscat. During the second day there was a panel composed of surgeons from the various areas discussing fractures of the long bones, presided over by Dr. R. J. BIGGAR, of the Bahrain Petroleum Company.

It is planned to hold the next meeting of the society at Kuwait in November, 1955.

Liverpool Medical Institution

On January 13 there was a discussion on "The Medical Education of Patient and Public." Dr. ROBERT KEMP, speaking of the medical education of the patient, emphasized the difficulties and drawbacks in providing patients with complete clinical information. However, there were a number of diseases, such as diabetes mellitus, pulmonary tuberculosis, duodenal ulcer, and epilepsy, where successful management depended almost entirely on the patient understanding his complaint. In such cases a formal educational approach to the patient was required, but the doctor had to be extremely selective both in regard to the material offered to the patient and in the type of illness in which education was undertaken. The approach to the patient differed somewhat from that of the profession itself to the general public.

Dr. I. HARVEY FLACK, editor of *Family Doctor*, said that there was a real need and a real demand for information and education on medical subjects. Ideally it should be met by the individual physician talking to the individual patient. In practice it very frequently had to be met in some other way. There were several possible methods, but one of the most important at the present time was by means of the magazine *Family Doctor*. Doctors had to be realistic and accept the fact that the public at large were not interested in health. They were interested in disease. They were interested in wonder drugs, miracle cures, and remarkable operations, not in the kind of health education that very often the profession wanted to get across to them. However, if one talked or wrote about a disease interest was attracted, and within that framework a good deal of valuable advice could be conveyed on prevention, healthy living, and the avoidance of infection. The only section of the public that was vitally interested in non-disease teaching were the young mothers, who were always anxious for information about the normal physiology of pregnancy and delivery, and about the care of infants, toddlers, and young children. There was a big field of interest here, and it was one that *Family Doctor* consistently tried to meet.

Devon and Exeter Medico-Chirurgical Society

On December 16, 1954, Dr. F. S. W. BRIMBLECOMBE spoke on some common disorders of childhood. Underfeeding was one, he said, but overfeeding very rare. If a baby failed to thrive it was wise to seek the cause in the baby rather than in the feed. Most of the causes of failure to thrive could now be recognized. Wrong feeding was one, but a rare one. Another was hiatus hernia : here the function of the cardiac sphincter was lost and the patient vomited from birth, especially when laid flat. The vomit contained mucus and sooner or later, owing to oesophageal ulceration, blood. Painful swallowing from the same cause produced anorexia, and occult bleeding might give rise to

anaemia. The diagnosis was a clinical one, for confirmation by barium *x*-ray examination was difficult. Feeds should be thickened and the baby sat upright. Occasional surgery was needed to free a stomach which had ridden up into the chest and become adherent.

Bottle-fed babies, apparently thriving, sometimes started to vomit at the third or fourth month, became costive, and developed unexplained pyrexias. Necropsy showed calcification in the proximal renal tubules which could be demonstrated radiologically during life. In these cases there was acidaemia with a urine constantly alkaline. This disorder, persistent renal acidosis, could be treated successfully with large doses of sodium bicarbonate. Another group, clinically similar, was due to hypercalcaemia of unknown origin, which was equally controllable by a calcium-free diet.

Preparations and Appliances

A HANDY OCCLUDER

Dr. G. J. ROMANES (London, W.1) writes: The use of a shield is convenient when assessing the visual acuity of each eye separately. Unless a special instrument is available the occlusion is usually carried out by covering each eye with any object to hand, such as a card or a piece of paper. This tends to give the patient an unfavourable impression. I have therefore evolved for this purpose a suitable occluder (see illustration), the features of which also enable it to fulfil several other functions.

The shape of the "head" allows it to rest comfortably on the bridge of the patient's nose, thereby occluding one eye for visual acuity assessment or for the cover test—that is, the unmasking of a latent squint. Both eyes can be covered simultaneously if desired—for example, during examination of pupil reactions. One side of the handle is

engraved with a millimetre scale enabling it to be used for interpupillary distance measurement. The end of the handle is rounded to allow it to be placed on the zygomatic bone, when a numerical idea of proptosis can be obtained from the scale. The reverse side of the handle incorporates a white spot 5 mm. in diameter, which can be used as a rough scotometry target or as a fixation point to help the patient look in a specific direction. The handle is tapered so that it can be utilized as a pointer to indicate the letters which are to be read on the test type. The whole instrument is designed so that its overall length enables it to fit into the accessory tray of a standard trial case.

I have used a similar occluder for some years now and find that it is a handy and time-saving accessory.

I should like to acknowledge the help which I have had in developing the accessory from the staff of Clement Clarke, Ltd., London, from whom the shield is now available.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

Fluoridation of Public Water Supplies

SIR,—We enter with great reluctance the controversy upon this topic mentioned by your correspondent, Dr. C. G. Dobbs (*Journal*, January 8, p. 105). Our reluctance arises partly from our profound ignorance both of the effects upon the human body of the chronic ingestion of low concentrations of fluorides and of the level of intake at which retention in the body occurs in different people. But we believe our ignorance is no greater than that of other members of our profession. Since one of us (D. C. W.) has worked upon the subject for some years, and since the other (H. M. S.) has been publicly and correctly reported as being opposed to fluoridation of public water supplies before further research is done, and has been consulted by Dr. Dobbs and many others, we feel it our duty to state very briefly our views.

It is well established that the ingestion of water containing 1 p.p.m. of fluoride decreases the incidence of dental caries in children up to the age of about 8 years; the effect on adults is much less certain. It is proposed that the public water supply of four places—Anglesey, Darlington, Kilmarnock, and Watford—should be fluoridated to the level of 1 p.p.m., presumably to increase the resistance of the teeth of children to caries during the period of their calcification. This will undoubtedly benefit the teeth of the children, but we do not believe it is without risk to the adults whose teeth may not be improved.

It can be argued that, because some 14 million persons in the U.S. are drinking public water supplies fluoridated to 1 p.p.m. without any demonstrated ill-health, the practice would be without risk to health in this country. But habits differ in the two countries. The two richest dietary sources of fluorine (according to McClure[1]) are sea foods, which as consumed may contain 5 to 15 p.p.m., and tea, of which the better grades contain 75 to 100 p.p.m. From these sources communities in the U.S. are likely to derive less fluorine than those in this country, and the consumption of tea may be highest among old people least able to excrete fluorides. It has been argued[2] that in this country adults who have arrived in West Hartlepool (with a water of natural fluoride content of 2 p.p.m.) or Maldon (5 p.p.m.) after the calcification of their permanent teeth "have drunk water containing 2–5 times the proposed concentration with no ill effect," but we have not seen the evidence for this statement. One of us (D. C. W.) took part in a study in which "severe dental fluorosis" appeared to be associated in some persons living in one village in Oxfordshire with a water supply having fluoride concentrations between 0.3 and 1.2 p.p.m. and in another village with a supply containing 0.8 p.p.m.[3] One of us (H. M. S.) quoted this work in a lecture to the Women Public Health Officers' Association last July to which wide publicity has unfortunately been given.

We believe the hazard from atmospheric pollution is greater in this country than in the U.S. Outbreaks of animal fluorosis have occurred from the use in industry in increasing amounts of compounds which naturally contain compounds of fluorine, such as "marine" coals and clays and the phosphatic iron ores; and there are at least 28 industrial processes in which added compounds of fluorine are used as a flux during manufacture. Such atmospheric pollution causing animal fluorosis in the neighbourhood of Stoke-on-Trent was the subject of recent questions in Parliament.

An adult eating ordinary foods (without sea food) with four cups of tea daily and drinking 1¼ litres of water containing 1 p.p.m. of fluoride would ingest about 2.2 mg. of fluoride daily. The level at which storage occurs in the body is not known: Machle, Scott, and Largent[4] found some storage on an intake of 2.0 mg. daily, and in a more recent study Largent[5] concluded that 1.36 mg. of fluoride daily for 45 weeks led in a healthy adult to storage of about 226 mg. during this period. In the elderly or in those with impaired renal function, storage is likely to be greater. More information is required.

Various authors, including one of us,[6 7] have concluded that the toxicity of fluorides depends upon the nutriture of the person concerned; calcium nutriture has been claimed to be particularly relevant.[8] This problem deserves further study. The United Kingdom Mission on the Fluoridation of Domestic Water Supplies[9] believed that "in theory any fluorine compound which will ionize in aqueous solution should have the same action on teeth." We take leave to doubt this. First, a solution of stannous fluoride has a greater influence in the rat on experimental dental caries than has a solution of sodium fluoride.[10] Secondly, it is not unreasonable to expect these two compounds to behave differently, at any rate in strong solution: a solution of stannous chloride, unlike a solution of sodium chloride, has a reducing power attributable to its solution containing much $SnCl_3^-$ anions,[11] and stannous fluoride might ionize similarly. So even on a simple chemical point there seems to be divergence of opinion. We do not even know how fluorides act on the teeth. There appears to be no protection against caries if they are given parenterally,[12] which would indicate that the effect is one following direct application to the surface of the tooth; but incorporation in dentifrices is alleged to be useless,[13] though others have claimed results from application to scaled and polished teeth. The effect on the oral flora deserves more attention. But we hasten to add that ignorance of how fluorides increase the resistance of the teeth of children to caries is no argument against their use.

Our argument is, in summary, that we do not consider it to be established beyond reasonable doubt that increasing the fluoride content of public water supplies to 1 p.p.m. is without risk to adults in this country. We consider that more research should first be done, which should include further studies upon storage in the body, particularly of the elderly.—We are, etc.,

H. M. SINCLAIR.
DAGMAR C. WILSON.

Oxford.

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Premedication of Children for Tonsillectomy

SIR,—I have read with interest the article and subsequent letters on this subject (*Journal*, December 11, 1954, p. 1397, and January 1, 1955, p. 44).

During the past five years I have tried the following combinations for guillotine tonsillectomy and adenoidectomy in children: (1) quinalbarbitone premedication followed by pure N_2O from a McKesson machine. This was associated with anoxia and occasional troublesome laryngeal spasm. (2) Syrup of chloral hydrate 5 gr. (0.32 g.) per stone (6.4 kg.) body weight for premedication followed by pure N_2O as above. No cases of laryngeal spasm and no untoward sequelae were seen, but as anoxia rather than anaesthesia formed the basis of the technique it was discontinued. (3) Premedication with atropine only followed by intravenous thiopentone 50 mg. per stone (6.4 kg.) body weight. Post-operative restlessness in the ward rendered this technique unpopular. (4) Premedication with syrup of chloral and atropine followed by ethyl chloride and ether or "neothyl." This proved too time-consuming for a long list. (5) Premedication with atropine only followed by vinyl ether, ethyl chloride, or trichlorethylene in the Oxford inhaler. Induction proved stormy and generally unpopular. (6) Premedication with atropine only followed by 50% cyclopropane in 50% oxygen and a trace of ether. As with the thiopentone series post-operative crying and restlessness ruled this out. (7) In a recent series of 100 children whose ages ranged from 5–10 years, 50 were premedicated with methylpentynol (2–4 teaspoonfuls) one hour pre-operatively and 50 with syrup of chloral 5 gr. (0.32 g.) per stone (6.4 kg.) body weight one hour pre-operatively. Induction was with pure N_2O, followed by N_2O and O_2 (10%) and a trace of trichlorethylene from a McKesson machine.

The nursing staff soon requested the abandonment of methylpentynol in favour of chloral because of pre-operative apprehension and post-operative restlessness. The only point in favour of methylpentynol would appear to be its effectiveness in removing the after-taste of syrup of chloral. While the ideal premedication and anaesthetic technique for guillotine tonsillectomy in children has yet to be found, syrup of chloral correctly given, followed by nitrous oxide, oxygen, and trichlorethylene, correctly administered from a McKesson machine, is at present, I think, the nearest approach to this ideal. My thanks are due to Dr. Kothary and Sister Beal, of the E.N.T. department, for their interest and co-operation.—I am, etc.,

Hull. W. N. ROLLASON.

Sickling and Malaria

SIR,—The *Journal* of November 13, 1954, has just arrived here and we have seen Dr. A. B. Raper's letter (p. 1162), with much of which we are in complete agreement. The problems presented by the hypothesis that there is an association between malaria and sickling are exceedingly complex and not capable of easy solution. We did not say that we had proved that sickling and malaria are not associated, but stated (*Journal*, September 11, 1954, p. 630): "There is no confirmation of the theory that there is an association of any magnitude, such as that suggested by Allison, between sickling and malaria" (*Journal*, February 6, 1954, p. 290).

The purpose of our survey among the Daruma and Kambe was to discover if very wide variations in sickling were accompanied by differences in malaria rates; we found none. Our determinations of sickling and malaria were done on the same individual. In Allison's work the samples investigated for malaria and sickling consisted of different individuals, in some instances in widely separated areas. Raper says that our numbers were small (302 Daruma and 220 Kambe), but we have since doubled them and have still found no correlation between sickling and parasite or spleen rates.

We are puzzled by Raper's reference to the mixture of adults and children that composed our sample. Since sickle-cell rates do not differ significantly between adults and children the validity of significance tests could not be affected. It is, of course, true that if there were a differential in malarial rates between sicklers and non-sicklers for children, but not for adults, a sample of children only would be more efficient for the purpose of determining associations. However, since our memorandum was published in the *Journal* we have selected a sample of children under six months and have again found no association between malaria and sickling rates. Results of this survey will be published later.

Numerous pitfalls are likely to be encountered in trying to correlate sickling and malaria, since any associations may be seriously influenced by a number of factors, such as population movements, genetic drift, and ecological changes. Any changes in gene frequencies, due to deaths from sickle-cell anaemia, would be very slow. We are, of course, aware that the survival of sickle-cell genes involves some selective advantage, but this advantage might equally well be due to either higher fertility or lower mortality.

We gather that Raper considers that the severity of malaria is less in sicklers than non-sicklers, since he found a lower parasite density in the former, and considers this to be one of the reasons for the persistence of high sickle-cell trait rates in various African tribes. At present we have very little evidence to contribute on this question of parasite density. However, in a survey that we have just completed among a very homogeneous group of Jaluo we have found no evidence that parasite density and sickling were associated. So far the numbers examined have been small and larger samples may reveal a different situation. This work is being continued.

We must emphasize again that we cannot accept any evidence of association where the homogeneity of the sample, with respect to the characteristics studied, is not proved. We have previously shown that sickle-cell trait rates vary very greatly from place to place, even within the same tribe, and this has now been confirmed by other workers. Parasite rates and densities as well as sickling vary even in quite small areas, and the chances of fortuitous associations are therefore high. Under such circumstances the assumptions necessary for tests of significance do not hold ; we gave an example of this in our memorandum. Our own experience, and that of others doing medical survey work in Africa, is that such fortuitous associations are common. Our reluctance to accept the results of tests of association at their face value is particularly strong when the sample is a mixture of tribes, as was Dr. Raper's. An association between parasite density and sickling, such as postulated by Raper, can only maintain the sickling gene by ultimately causing a differential mortality. Even if parasite density can be shown to be lower in sicklers, it remains to be proved that this is associated with a lower death rate in the *untreated* patient.

Further, is parasite density really a measure of malarial severity in African natives ? The pyrogenic density or threshold is a factor of great variability in falciparum malaria in immunes as well as non-immunes. Severe clinical symptoms may occur with densities as low as ten parasites per cubic mm., and in other cases large numbers of parasites may be present and give rise to only the slightest illness. The presence of parasites in blood and particularly their density is dependent on a number of complex and, so far, little understood factors. No single entity, such as sickling or immunity, can be regarded as paramount. The high traumatic fragility of blackwater fever red cells is one of the factors that contribute to the scarcity of parasites in this disease. Similarly, in sickle-cell trait the traumatic fragility of the parasitized red cells may result in their being destroyed at an early stage of infection and thus reduce parasite density both by direct destruction of red cells and by limiting the number of parasites that undergo schizogony ; we do not, at present, know whether there is a more intense destruction of red cells in cases of malaria in sicklers than in malaria in non-sicklers. This is a question that can only be answered by careful blood studies particularly of pigment metabolism.

There may be a "negative association" between sickling and mortality from malaria, but we cannot agree that it has yet been proved. We are not altogether convinced that an association between *parasite density* and sickling has been demonstrated, but even if it has we cannot agree that parasite density and mortality from malaria can be equated. —We are, etc.,

HENRY FOY. G. L. TIMMS.
W. BRASS. R. A. MOORE.

Nairobi, Kenya.

Medical Education

SIR,—I was interested to read Professor L. J. Witts's review of the *Proceedings of the First World Conference on Medical Education* (*Journal*, October 9, 1954, p. 853) which has recently arrived here. As Professor Witts says, there are too many thoughts and not enough experiments in medical education. It is heartening to read of the various recent work in medical education in the United States of America.

In Queensland, we have departed from the usual system of written examinations in the final year, and have introduced oral and clinical examinations only ; written examinations have been included at the end of the fifth year. This was recommended by Sir William Osler long ago. At the present time, there is an urgent need to make better provision for the requirements for entry to medical schools. All students should have a knowledge of the arts, sciences, and sociological subjects, and thereby would have a good basis for general education.

Now that arrangements for the academic training of those who are going to be specialists of one sort or another follow set lines, the undergraduate curriculum may be considered. Sir A. L. Mudaliar, who presided over the section on the "Aims and Content of the Medical Curriculum," has put the case quite well. There is need for a reduction in the amount of anatomy and physiology taught to undergraduates, and, as Sir A. L. Mudaliar said, "It would be well if the student would occasionally look into the hospital ward, and be shown what was normal and what was abnormal. . . ." Similarly with pathology, "the pathologist would walk into the wards, and tell the students exactly what he felt about pathological conditions. . . ." Years ago Lord Moynihan stressed the importance of the "pathology of the living," and quite recently Lord Horder and Sir Heneage Ogilvie have emphasized that medical students are not going to be professors of these subjects, but should be able to recognize anatomical and physiological conditions as met with in general practice.

The *Proceedings of the Conference on Medical Education* will require careful study, and, as has been said, Dr. Clegg and his colleagues have done well to produce the book so quickly. It seems to me, however, that unless arrangements can be made for universities to exchange views on their efforts to produce a better curriculum from time to time the full benefit of the work of the conference will not be realized.—I am, etc.,

Brisbane, Australia. E. S. MEYERS.

Reading and Writing about New Diseases

SIR,—For those of us who spend a few week-end hours in trying to keep up to date in new medical discoveries about which we know nothing, it would be most helpful if a preliminary paragraph introduced us as simply as possible to the subject. For instance, I have to-day battled with articles on phenylketonuria (*Journal*, January 8, p. 57) and byssinosis (p. 65) without ever getting my ignorant feet on the ground for a good start in understanding them. I have always told myself and my juniors to kick off with a simple explanatory paragraph relating the past to the new when writing about specialized novelties. I hope you, Sir, will insist on this when your authors tend to neglect it.—I am, etc.,

London, W.1. R. D. LAWRENCE.

Talc Pneumoconiosis

SIR,—I read with much interest the letter from Dr. David Weitzman (*Journal*, January 8, p. 104). The atomic lattice structure of talc is such as to render its inhalation in powder form unlikely to cause a true pulmonary silicosis. Nevertheless, some unsatisfied valencies may be found at the edges of the platelets in freshly powdered talc, just as they are found at the fractured ends of freshly powdered asbestos fibres, and may possibly produce similar results. For a recent discussion on the chemical surface activities of silica and the silicates your readers are referred to *Tubercle* for September, 1953.[1]—I am, etc.,

Cork. PATRICK HEFFERNAN.

REFERENCE

[1] *Tubercle*, 1953, 34, 246.

Treatment of Papilloma of Bladder

SIR,—I have just read with very considerable interest the article by Dr. Frank Ellis and Mr. R. Oliver on the above subject (*Journal*, January 15, p. 136). I do not wish to appear in any way critical, as the treatment of papillomatosis of the bladder has always been notoriously unsatisfactory, and the case described by the authors would have otherwise come to total cystectomy, an operation which must be regarded as a last resort, for, no matter what steps are taken to minimize the subsequent disability, the patient will remain a potential invalid for life.

I feel, however, that one statement should not be allowed to pass without comment, as it may give an entirely false impression of the results of the more common treatment for vesical papilloma. This reads : "The treatment of these lesions by cysto-diathermy has for many years been the method of choice, but the results of such treatment are difficult to find. It is certain, however, that a number of patients develop incurable malignant disease after many years' cystoscopy and diathermy." During the past ten years I have been privileged to see a large number of cases of vesical papilloma treated with cysto-diathermy, and no

single case which was diagnosed clinically as a simple papilloma has later developed characteristics of malignancy. Furthermore, I have seen several cases which had the cystoscopic appearance of malignancy, but in whom radical surgery was contraindicated. Following treatment with cysto-diathermy the growths have regressed permanently, and in some instances have disappeared. Cysto-diathermy is a treatment for these lesions which has, in my humble opinion, successfully stood the test of time, and must not be hastily abandoned for a far more hazardous procedure. —I am, etc.,

Slough. ROBIN BURKITT.

Epithelioma Due to Tar Ointment

SIR,—We are grateful to Mr. W. H. Bond (*Journal*, November 20, 1954, p. 1226) and Dr. R. E. W. Fisher (*Journal*, December 4, p. 1355) for their comments on our case report (*Journal*, November 6, p. 1089). In his letter Mr. Bond states that "with the solitary exception of the histological report *all the reported features are convincingly those of molluscum sebaceum*" (the italics are ours). In their original description of molluscum sebaceum MacCormac and Scarff[1] list six features of the condition. They are: (1) The lesion develops rapidly to its maximum in four to six weeks and then remains stationary. (2) In no case has more than one lesion been present. (3) The middle area of the face is the site of election. (4) The lesion is usually of firm consistency but may show central degeneration. (5) In all cases the patients were of mature age—generally over 60. (6) Both sexes were affected. In a further series reported by Beare[2] only seven out of seventy-six were below the age of 35; the majority were over 50. In other respects the features of Beare's cases conformed to the original description. Mr. Bond puts forward (1) the age of our patient—that is, 31; (2) the apparent absence of other pre-malignant or benign tar tumours on other parts of the body treated with tar (when there were in fact several tar mollusca also present in the treated area); and (3) the appearance of a tumour within a patch of psoriasis, as convincing evidence of molluscum sebaceum. We are quite unable to agree with this view. The short exposure to tar is unusual, but we believe that this feature was compensated for by the patient persisting in its use for a considerable period after signs of tar intolerance had appeared. Incidentally, we are not aware that tar is an aetiological factor in molluscum sebaceum.

Savatard[3] commented on MacCormac and Scarff's cases that the features were those of the well-known button epithelioma, a criticism almost the reverse of what Mr. Bond says about our case. In their reply MacCormac and Scarff[4] emphasized one point very clearly. They said ". . . the appearances of the section of the growth are much more nearly matters of fact, and we confess, with all respect, that we can find nothing in those appearances which suggest epithelioma or rodent ulcer either in the slide or the photograph." We have re-examined the sections in our case. The appearances are those of a well-differentiated, keratinizing, squamous carcinoma of low-grade malignancy. There is a hyperplasia of the epithelium around the tumour—a feature not found in molluscum sebaceum.

With respect to Dr. Fisher's comments we agree that tar epithelioma can occur without the presence of other lesions and may even appear in a man who has long since given up working with tar. It was not our intention to suggest that tar acne is an *essential* precursor of tar epithelioma. On the other hand, it is the *repeated* stimulation of the skin which results in the malignant change. Other signs of such stimulation or irritation are also very often present. The late onset of epithelioma due to tar in industrial workers, often at a time when they are no longer exposed to tar, may account for tar acne being absent at that time. In passing we would remark that the failure to make a diagnosis of cancer because of the absence of tar acne does not commend the clinical acumen of the examining physician to us. Surely a cancer lesion should be diagnosed or at least suspected on its clinical appearance and not on ancillary findings.

We question whether in fact industrial workers do rub tar into their skin any more than our patient did. The latter used a very strong ointment made up in emulsifying base, which is expressly designed to secure quick and deep penetration of the skin. This occurs particularly around the hair follicles. Anyone who has used tar ointment extensively knows that this may sometimes produce folliculitis or tar acne, and this is an indication for withdrawal of tar therapy.—We are, etc.,

J. O'D. ALEXANDER.
Glasgow, C.4. K. I. MACROSSON.

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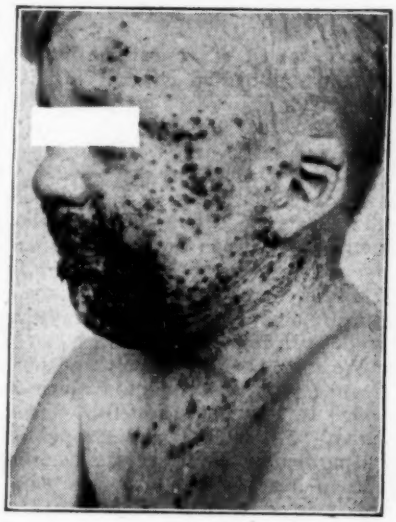
Kaposi's Varicelliform Eruption

SIR,—I have been prompted to protest against the title, "A Case of Kaposi's Varicelliform Eruption (Accidental Vaccinia)" reported by Dr. A. Fry (*Journal*, January 1, p. 28). The case reported undoubtedly refers to accidental infection with vaccinia, but the lesions described are those well recognized as generalized vaccinia. Dermatologists reserve the title of "Kaposi's varicelliform eruption" for the alarming eruption often associated with severe constitutional disturbance and high fever which occurs when a case of infected eczema also becomes infected with the virus of herpes simplex or vaccinia. In these cases the virus infection is usually a primary one, but it may not be, because recurrences of Kaposi's varicelliform eruption have been reported.



When a virus has been identified most dermatologists would prefer a diagnosis of eczema herpeticum or eczema vaccinatum, but the clinical picture of Kaposi's eruption, as illustrated by the above photograph, is nothing like that of accidental vaccinia. It is probable that a spreading factor such as hyaluronidase, present in infected eczema, accounts for the rapid local spread of the virus in cases of Kaposi's varicelliform eruption. Although the vaccinial cases of this disease may well be described as accidental they can readily be distinguished from vaccinia.—I am, etc.,

London, W.1. REGINALD T. BRAIN.

A Case of Human Anthrax

SIR,—The authors Mr. M. F. Hunt and Dr. P. Cook (*Journal*, January 8, p. 87) and medical practitioner in charge of this case are to be congratulated for their early diagnosis, treatment, and subsequent investigations. It would be of interest to learn if "bone meal" was a constituent of the "animal manure," and, if not, had the inoculating insect access to this substance in a neighbour's garden. In recent years several writers[1–4] have stressed bone meal as a source of *B. anthracis*, as frequently the dried bones are imported from Asiatic countries where anthrax is endemic.—I am, etc.,

London, W.1. IAN MARTIN-SCOTT.

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Low Backache

SIR,—The methods of treating disk lesions advocated by Dr. James Cyriax (*Journal*, January 15, p. 140) are, he says, "based on logic." Logical thinking in medicine must always be followed amongst others by the question: Does it work in practice?

Cyriax suggests with all his strength that his methods do work: in one instance "immediate manipulative reduction succeeds in 99% of all cases," and "this simple manœuvre prevents the later onset of brachial neuritis," presumably in 99% of cases. Again, referring to the dorsal spine, he says, "A protrusion irreducible by manipulation during manual traction is a rarity," and for the lumbar region his summary is, "In backache manipulative reduction, without anaesthetic, is usually successful," although there is a footnote stating that "the pain seldom returns to anything like its previous intensity." These manipulations have been carried out in Dr. Cyriax's department "with manifest success," and physiotherapists, he says, can be trained to do them, "as our experience at St. Thomas's Hospital proves."

The problem set by painful disability from disk degeneration and protrusion is very difficult and troublesome, and we would be inclined to agree with Cyriax's estimate of 20,000 to 50,000 individuals disabled from their work at any one time by these disorders.

If Cyriax really has the answer to both cause and treatment, as his article is meant to suggest, we must learn from him and train doctors and physiotherapists to carry out his methods. But can he really produce the goods? Can he prove that his rather elaborate technique, including manipulation, is more rapidly and surely successful than the established methods of exercise therapy which have always been productive of moderate success?

We must examine closely and objectively any method of treatment for any condition which claims "99% of cures," which is "usually successful," and in which "failure is a rarity." So far Dr. Cyriax has published many statements[1–5] but no figures. Many of the syndromes which he treats are apt to resolve spontaneously whether the patients are manipulated or not, and in two or three weeks. Gradual improvement during a series of manipulations at daily or weekly intervals cannot easily be disentangled from the probability of spontaneous cure. Will Cyriax produce rather more concrete evidence of the success of his methods so as to draw them more convincingly from the morass of palliative osteopathy? Will his colleagues at St. Thomas's Hospital or in the specialty of physical medicine support his claims for outstanding success?

Finally, expert medical opinion is at present such that a patient might argue much more successfully in a court of law that a manipulation had done harm than that recovery had been delayed by a negligent failure to manipulate at all.—We are, etc.,

W. D. COLTART.
H. OSMOND-CLARKE.

London, N.W.1.

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2 *British Medical Journal*, 1948, **2**, 251.
3 *Osteopathy and Manipulation*, 1949. Lockwood, London.
4 *British Medical Journal*, 1950, **2**, 1434.
5 *Orthopaedic Medicine*, 1954. Cassell, London.

SIR,—All those concerned with the treatment of low backache should make themselves acquainted with the important report published by Romanus.[1] It is based on a careful study of 117 male patients. His data make it quite clear that the bone changes not only of the spine and pelvic girdle but also of more peripheral parts are the result of chronic prostato-vesiculitis. To establish such a relationship reliable means of demonstrating the presence of prostatic infection must be employed. Certainly the examination of a single prostatic smear is inadequate. It is astonishing at first to find that a prostate which feels quite normal on rectal examination can show granulomata in the prostatic sinuses or on the verumontanum. To me such findings are evidence of badly draining infective foci—the proof of which can often be shown by improvement in the urinary symptoms (commonly chronic frequency) and backache. In women the regular use of the urethroscope for more than 20 years on all those with disturbances of micturition has revealed to me concealed chronic inflammatory foci in the posterior urethra, often in the presence of a crystal-clear and sterile urine. Treatment of these foci in many cases will cure or improve the accompanying backache.

The appropriate treatment of cervical erosion, and chronic vaginitis, will often have the same effect. Improvement from treatment, however, is not to be expected when the lower urinary tract condition has progressed to an established *Bact. coli* infection or fibrosis of the bladder neck, and chronic changes in the upper urinary tract. The *x*-ray films of the pelvis and spine which the urologist so commonly examines are constantly reminding him of the presence of osteoarthritic changes in chronic urinary cases.—I am, etc.,

H. P. WINSBURY-WHITE.

London, W.1.

REFERENCE

1 *Pelvo-Spondylitis Ossificans in the Male, and Genito-Urinary Infection*, 1953. Stockholm.

SIR,—Mr. John Charnley's views on low backache (*Journal*, January 15, p. 163) command respect, but it is doubtful whether they can be adopted as "facts." I find it difficult to accept some of his contentions, and trust I will be forgiven if I examine some of them from the point of view of a peripheral general surgeon who is not exempt from some responsibility for this skeletal disability.

Surely consultants employed by a National Health Service must think twice about withholding treatment unless "there is unanimity that the results . . . are worth the financial outlay." Most sensible patients approach the doctor in order to have some incommoding symptom removed or mitigated. They are not concerned with abstract (though useful) concepts such as "significant organic causes," "scientifically sound rationale of treatment," or even "diagnosis." The docker or his wife with backache wants the ache removed. If it is not removed the patient may, in spite of consultations, radiographs, corsets, or even "reassurance without treatment," get the idea that nothing has been done. He may even become "impossible to reassure" and finally resort to a quack who may cure him. I doubt whether, in the present state of knowledge, it is practicable to resolve pain into "organic and functional elements." As far as I am concerned persistent pain is a real thing, even when I cannot see, feel, or *x*-ray the aetiological lesion. A significant lesion may be physically minute. Mr. Charnley's colleague, Professor Kellgren,[1] demonstrated that the injection of 0.1 ml. of hypertonic saline into an appropriate skeletal structure could cause severe widespread segmental pain. It is easy to conceive of natural lesions so small causing persistent symptoms. The prolapsed disk is a common small yet significant lesion which escaped the attention of our profession for generations. Who can demonstrate the lesion of tennis elbow or facial neuralgia?

The history of our art is rich in examples of skeletal pains ignored by the orthodox only to be successfully treated by the unorthodox.[2–7] Rational physiotherapy is more economical than irrational. Dr. L. S. Calvert (*Journal*, December 18, 1954, p. 1488) rightly drew attention to methods of physical medicine long exploited in the back street and by osteopaths, but now greatly improved and clarified in terms of orthodox medicine by Dr. Cyriax.[8] Cyriax has described rational and refined methods of examining patients with aches and pains and has based on them a reasonable system of physical medicine[9] of which the main ingredients are deep friction, manipulation, and traction, accurately applied to the site from which the pain is thought to emanate. *En passant* he has dealt severe blows at that honoured clinical nonentity "fibrositis." In the limited compass of his writings he has described his methods without producing evidence strong enough to compel acceptance of them, but by testing them it is easy to see that they produce benefits in many cases not amenable to traditional physiotherapy, much of which

justifies Mr. Charnley's description of "six weeks' quackery." It is conceded that there is a considerable residuum of incurables, but I share Mr. Charnley's hopes for the future.

The golden age of surgery was preoccupied with visceral disease, and most classical researches on pain, from McKenzie to Morley, were concerned with visceral pain. It is possible that many genuine pains due to obscure skeletal lesions have, in the past, been brushed aside as "not due to significant lesions" or "functional." I encounter many pains which I cannot diagnose or cure, but consider it salutary to attribute this to the limitations of my knowledge and methods. The researches of Kellgren and Lewis[10] have thrown new light on the mechanisms of somatic pain, and I submit that Dr. Cyriax has significantly illuminated the clinical exploitation of this knowledge. We must test, not reject out of hand, all promising ideas. The great Paget,[2] in this connexion, said, "*Fas est ab hoste docere.*" It is even better to learn from the successes of our less orthodox colleagues. To do so may protect the profession from some public criticism as in the case of Sir Herbert Barker.[7]—I am, etc.,

Hull. J. CLAPHAM COATES.

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7 *The Times*, July 22, 1950. Obituary on Sir Herbert Barker.
8 *Textbook of Orthopaedic Medicine*, 1954, vol. 1. Cassell, London.
9 Ibid., 1950, vol. 2. Cassell, London.
10 *Clin. Sci.*, 1939, **4**, 47.

SIR,—Dr. James Cyriax (*Journal*, January 15, p. 140) performs a great service in pointing out in his paper the value of manipulative procedures in the treatment of low backache, but he does not disclose his diagnostic criteria for the diagnosis of spinal disk lesions.

Harris and McNab,[1] of Toronto, pointed out in 1952 that only a small percentage of the 123 patients then examined at necropsy had disk lesions and that other anatomical deformities and degenerations were probably the basis of both the patients' backache and root pain; thus we may assume that the majority of patients attending the practitioner and hospital clinics will have symptoms not related to disk derangement. Whereas a percentage of the latter will also respond favourably to competent manipulation, a large number would not only make no improvement but may indeed be made far worse by this form of treatment, especially if attempted by the inexperienced.

Prior to advocating widespread manipulative therapy in this country for all cases of backache, would a greater service not be rendered, Sir, by the clarification of the various diagnostic procedures required to make a correct diagnosis in the many sufferers from backache? In low back pain, more than in any other condition, accurate diagnosis is the only safe starting-point for successful treatment.—I am, etc.,

London, S.E.1. P. HUME KENDALL.

REFERENCE

1 *J. Bone Jt Surg.*, 1954, **36B**, 304.

SIR,—As a general physician often concerned, and frequently confounded, by the chronic or recurrent lumbar backache problem, I view the diametrically opposed opinions of Dr. James Cyriax (*Journal*, January 15, p. 140) and Mr. John Charnley (p. 163) with baffled and amused wonderment.

As I see it, Dr. Cyriax (the sanguine) considers the majority of chronic backache sufferers to have small posterior disk protrusions, easily cured by manipulation (delegated to the physiotherapist), while Mr. Charnley (the pessimist) considers the vast majority have either no physical disease (presumably psychoneurotics) or have a physical disease he cannot diagnose or treat, and so he reassures them. Where does the truth lie? Has Mr. Charnley previously adopted Dr. Cyriax's methods and abandoned them as ineffective, too reminiscent of osteopathic practice, or as pure psychotherapy, to be classed with E.C.T.? Is the apparent success of Dr. Cyriax really due to the reduction of existing disk protrusions, is it a form of psychotherapy, or perhaps the patients, once subjected to manipulation without anaesthesia, are wise enough to keep their backaches strictly to themselves?

Quite seriously, the low backache problem remains an unsolved mystery in perhaps half the cases we see, for we are uncertain of the diagnosis, and therefore lack confidence in the treatment and management. I write this letter in the hope that the protagonists will take up the cudgels once more in the vertebral no-man's-land, and perhaps, who knows, we shall find that there is a wider common agreement than I imagine.—I am, etc.,

Warrington. DENIS M. FREEMAN.

SIR,—I have a great admiration for Mr. John Charnley as an inventor of ingenious orthopaedic instruments, but I am considerably shaken by his conception of a "humane and sympathetic Health Service" (*Journal*, January 15, p. 163). It is, of course, apparent that Mr. Charnley has himself had neither sciatica nor lumbago, but if and when his good fortune ceases I wonder how he will react to "reassurance without treatment." I am sure that he is "an intelligent, decent, and sensible" person, but will "sympathetic explanation" be any comfort to him in his suffering? No. He will probably "hand-pick" himself into the select residue who have their spines fused.

Was it by design or chance, Sir, that in the same issue as Mr. Charnley's letter appeared Dr. Cyriax's article (p. 140)? Their mutual contradictions as regards the efficacy of physiotherapy made intriguing and amusing reading. One cannot dismiss Dr. Cyriax's results by the trite phrase "*ad lib.* physiotherapy" and one suspects that Mr. Charnley is begging a big question when he implies that "organic" backache is a matter of "collagen degeneration." It might be an idea for the British Orthopaedic Association to invite Dr. Cyriax to be a member of the panel at their next brains trust. It would certainly dispel the "dog-in-the-manger" attitude which undoubtedly exists, and might even persuade Mr. Charnley to stop bellyaching about the "backaching multitudes."—I am, etc.,

Loughton, Essex. DONALD V. BATEMAN.

SIR,—As one who has had the opportunity of seeing Dr. James Cyriax's work and hearing the testimony of some of his patients, I feel that its value cannot be doubted, particularly in his insistence on accurate diagnosis before physiotherapy treatment is instituted and in his bringing to our notice the all-too-long-neglected forms of manipulative and traction therapy, which have reached a high standard in his hands. However, I must say that I believe equally firmly that he does his cause a great disservice in his over-simplification of the various intervertebral disk lesions and his insistence that the essence of their treatment is the reduction of the protrusion in all cases. Nothing in fact in a study of the anatomy and pathology of the intervertebral disks, particularly in relation to the effects of ageing on their physiology, lends support to this simple mechanistic theory, and a comparison of the lesion with that of a displaced meniscus of the knee-joint, as Dr. Cyriax has done,[1] is entirely misleading. I have already reviewed some of the reasons for this mistrust,[2] and subsequent work on dissections of the cervical and lumbar regions has merely served to confirm this.

Our ignorance of the exact cause of the symptomatology associated with the various types of spinal disk lesions is profound, but I believe there are reasonable grounds for rejecting the simple organ-stop approach, that is to say, disk out giving a *vox radiculus* or *vox ligamentus* and, alternatively, disk in giving perfect comfort. The spread of this conception is making the work of orthopaedic surgeons interested particularly in this problem difficult. I mention orthopaedic surgeons in order to refute emphatically the

implication of the quotations which precede Dr. Cyriax's article (*Journal*, January 15, p. 140), and to assure him that there are many surgeons concerned with this problem who could have given a great many suggestions for coping with the vast number.

Furthermore, I feel that he is playing also into the hands of the very people whom I know he is anxious to defeat—namely, the unqualified manipulator. For, now this mechanistic concept of the prolapsed disk and its treatment has been given an aura of medical respectability, many doctors find it difficult to understand why they should not advise their patients to attend lay manipulators who are ready to claim they can reduce three or four disks at each session, whether in fact there is any reason to suppose they are displaced or not.—I am, etc.,

Wellington, Salop. G. K. ROSE.

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[2] *Lancet*, 1954, **1**, 1143.

Heel Comfort

SIR,—I have lately had great success with an idea for treating painful heels in crippled or bed-ridden patients. A strip of "latex foam," 8 in. (20.4 cm.) by about 18 in. (45.7 cm.), put upside down between the sheets at the foot of the bed takes the heel or both heels in the squares, the front of which must be cut away. The depth of the squares varies from about 1 in. (2.5 cm.) to about 6 in. (15.2 cm.). I have used 2 in. (5.1 cm.), but where there is a risk of foot-drop the 6 in. (15.2 cm.) depth would help. The comfort has not only been to the heel, but one arthritic who has a pin in the neck of the femur has lost her pain in the hip and back also. She carries the cushion with her to sit on as well.—I am, etc.,

New Milton. MARY G. H. EDWARDS.

Intramuscular Iron

SIR,—We would correct the impression given in the letter from Drs. Sheila T. Callender and Mary D. Smith (*Journal*, December 18, 1954, p. 1487) that we found the preparation of intramuscular iron to be innocuous when given intravenously. During certain observations made[1] two anaemic and two healthy subjects were given 5 ml. of the iron preparation, first intramuscularly and then, after an interval, intravenously, but "the experiment was then abandoned because the patients developed moderate reactions after intravenous injections." The intravenous use of this preparation is not without risks and in our opinion is unnecessary.

With regard to Dr. Callender's and Dr. Smith's experience of reactions in four patients after injection of the preparation, two in fact received the iron intravenously, and of the two patients given iron intramuscularly one was already known to be of allergic disposition. We found no reactions in the 40 patients treated with an experimental preparation of intramuscular iron, but since publication of our results we have found one batch of iron preparation which, when given intramuscularly to two patients, produced malaise, pyrexia, and shivering. When a further batch of "imferon" was substituted no reaction of any kind was noted in either case to the same dose given intramuscularly. It is evident that certain batches of the iron preparation contain a pyrogenic substance.

The difficulties of assessing "reactions" can be judged from the following case report: A woman aged 64 was admitted to the dermatological department, Royal Infirmary, Sheffield, for treatment of psoriasis, and while in the ward an iron-deficiency anaemia was discovered. After oral treatment with ferrous sulphate and ferrous gluconate severe diarrhoea ensued, so imferon therapy was commenced. After two intramuscular injections of 5 ml. severe diarrhoea and some pyrexia occurred. Two possibilities were considered: (*a*) this was an allergic response to iron, (*b*) some iron was being excreted in the faeces—contrary to accepted concepts of iron metabolism. A further injection of 5 ml. of the same batch of imferon caused no diarrhoea and an iron balance experiment revealed no increase of faecal iron excretion after the injection. The diarrhoea was evidently coincidental to the intramuscular iron therapy.

Further experience is necessary to evaluate the frequency of reactions after intramuscular iron, particularly with regard to the occurrence of pyrogenic batches, and we feel that the published data indicate a useful but restricted place in therapeutics for the preparation, and the occasional occurrence of reactions should not deter from its use in selected cases.—We are, etc.,

Sheffield, 6. I. MCLEAN BAIRD. D. A. PODMORE.

REFERENCE

[1] Baird, I. McL., and Podmore, D. A. (1954). *Lancet*, **2**, 942.

Trichomonal Vaginitis

SIR,—I was interested in the article by Drs. S. R. M. Bushby, R. D. Catterall, and Mary Williamson (*Journal*, January 8, p. 78) reporting the results of a clinical trial of yet another drug in the treatment of trichomonal vaginitis. I am prompted to write a few brief comments based on my experience of treating a large number of cases of vaginal discharge.

Over 40% of cases complaining primarily of vaginal discharge are due to infestation with *T. vaginalis*. No age is immune; the disease has been observed in pre-puberty and after the menopause, but is more common during the reproductive years, especially in pregnancy. Clinical diagnosis in the acute phase is easy but should be confirmed by examination under the microscope; swabs are unreliable; pipette specimens examined fresh are essential. Ideally no case should be treated without demonstrating the organism; a week's supply of vaginal pessaries for "leucorrhoea," though giving temporary symptomatic relief, is a potent factor in the production of chronic cases. Acetarsol gives the highest percentage of cures, but it must be realized that to be effective the treatment should start with an initial insertion of 12 tablets and then daily insertions of one for at least 8 weeks, including during the menstrual periods. Weekly vaginal paintings with 2% gentian violet in spirit is a useful adjunct, and all marital relations should be "protected" by a condom for a minimum period of 6 months. The relapse rate with this routine is very small and a further course of treatment is usually effective.—I am, etc.,

Cambridge. OSWALD LLOYD.

Sequelae of Erythroblastosis

SIR,—In your leading article (*Journal*, January 1, p. 35) you drew attention to the problems of kernicterus. Your suggestion that experimental kernicterus has been produced by bilirubin infusion is, however, a little misleading. Day[1] exposed cerebral tissues to *x* rays and then demonstrated that the infused pigment stained the damaged portions. Kuster and Krings[2] appear to have produced little more than a generalized yellow staining of the brain, but in Kuster's original paper[3] reference is made to some staining of the basal nuclei when the serum bilirubin concentration rose to 75–100 mg. per 100 ml. In humans the characteristic pathological and clinical feature of the disease is the tendency for certain nuclear masses to be picked out and damaged, and the only experimental work which comes anywhere near achieving this state of affairs is that of Eyquem.[4] By the injection of anti-cat red cell serum into kittens he produced an acute haemolytic anaemia with jaundice, acute neurological disturbance, and staining of the basal ganglia. Although acceptable as experimental kernicterus, this work does not give any further clue to the pathological processes in the brain; Eyquem himself concluded that a local antigen-antibody reaction was responsible and that bile-staining was secondary. The widespread incidence of kernicterus without any evidence of an antigen-antibody reaction now makes this interpretation untenable; the typical clinical and pathological changes have been reported in prematurity, in acholuric jaundice,[5,6] and in "congenital familial non-haemolytic jaundice."[7]

There is strong circumstantial evidence pointing to bilirubin itself as the toxic agent, and a rational explanation of the high incidence of kernicterus among premature infants can be put forward when it is remembered that bilirubin levels in the blood of the newborn are inversely related to maturity[8] and that the "blood–C.S.F. barrier" is less effective in premature than in full-term infants.[9]

Despite these clinical observations, experimental evidence is lacking. Although successful attempts have been made to demonstrate the toxic effects of "bile" in the young animal[10] they cannot be accepted as incriminating bilirubin itself, as the material used was "concentrated bile" and not the pure pigment. To settle the role of pigments in the pathology of kernicterus it would seem necessary to show (1) that pure indirect-reacting bilirubin is toxic to the central nervous system and that if introduced into the cerebrospinal fluid it shows "special affinity" for the nuclear masses; and (2) that in both experimental and clinical kernicterus there are special circumstances which permit bilirubin to reach the cerebrospinal fluid in large amounts. It should not be long before evidence on these points is forthcoming.
—We are, etc.,

D. C. A. BEVIS.
R. M. FORRESTER.

Urmston, near Manchester.

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Changes in the Mental Health Services

SIR,—The recent correspondence on mental observation wards has shown that opinion is divided regarding whether or not their continuance in the future is desirable. Most of your correspondents have, no doubt, been arguing from their knowledge of such units in their own localities, and as there is no clear definition of what constitutes a "mental observation ward" the arguments are tending to become confused.

If by the term "mental observation ward" one is to understand a ward in a general hospital where patients on 3- and 14-day orders are admitted, for the purpose of deciding whether or not treatment in a psychiatric hospital is desirable (as voluntary, temporary, or certified patients as the case may be), or whether the patient can be discharged home, I can see no justification whatever for their existence—except possibly in the very largest centres of population where conditions are somewhat different. In any lesser centre the unit will inevitably be too small to allow of proper classification of the patients, and too small also to be administered and staffed efficiently as an economic proposition. Equally important objections are (*a*) that the patients requiring admission to the psychiatric hospital at the end of their "observation" will usually have to suffer a change of doctor as well as of nursing staff, and (*b*) that they will feel that they have failed to make the grade, and will go to the psychiatric hospital with an unnecessary feeling of shame.

On the other hand, it is generally agreed that psychiatric wards of some kind in general hospitals are desirable: such wards, however, should be for the most minor degrees of mental illness (including psychosomatic conditions) for whom admission to a psychiatric hospital might be difficult to arrange. If the Royal Commission recommends that the 3- and 14-day order procedure shall continue, the best place, in my experience, to which patients on such orders should be admitted, is the psychiatric hospital. Apart from many obvious advantages of such an arrangement, it is found in practice that the need for certification hardly ever arises, as is seen from the following figures for a provincial psychiatric hospital for the year 1954.

Admissions			Disposal						
3-day	14-day	Total	Vol.	Temp.	Cert.	Died	Home	To Other Hospital	Otherwise Disposed of
40	106	146	107	6	8	5	15	3	2

—I am, etc.,

Ivybridge, South Devon. FRANCIS PILKINGTON.

Cardiac Asthma

SIR,—Congratulations to the *British Medical Journal* for once more publishing outstanding articles which are of so much benefit to general practitioners. I read with great interest the first of the new series written by Dr. J. G. M. Hamilton, of Edinburgh, on cardiac asthma (*Journal*, January 1, p. 39). This condition is not a common emergency seen in general practice, but is one of the most satisfactory to treat. The use of morphine as quoted by the distinguished author as the only treatment in severe cases cannot be overemphasized. May I describe an outstanding case which I attended five months ago to substantiate this statement?

The patient, a man aged 67, had to my knowledge suffered from aortic regurgitation and cardiac hypertrophy for the past six years, having had attacks of breathlessness and been treated with digitalis and aminophylline. I was called to see him in the early hours of the morning and found him sitting by the open door very dyspnoeic. On examination, the patient was cold, clammy, and cyanosed. Neck veins were engorged and frothy sputum exuded from his mouth. Pulse was impossible to estimate, pupils sluggish, and he could not speak. His condition was truly grave.

Owing to the obesity of the patient—18 st. (114.4 kg.)—and urgency of the condition, ¼ gr. (32 mg.) morphine was given intramuscularly. Within 25 minutes a dramatic change occurred. The cyanosis and coldness of the skin disappeared although a slight degree of breathlessness remained. Pulse was now countable too. Mental outlook was normal, and he spoke intelligently. One hour later he insisted on returning to bed, and aided by a friend walked to his bedroom.
—I am, etc.,

Leicester. ERIC LODGE.

Treatment of Bronchial Obstruction

SIR,—The reason why the odds given by Dr. J. W. Trevan in his letter (*Journal*, January 8, p. 103) cannot be used to measure our degree of belief in the relative inefficacy of calcium chloride (*Journal*, December 11, 1954, p. 1388) is that, although our patients were chosen "at random," the trial was not concluded at a predetermined point (which would again be "at random" as far as the results were concerned), but at a point determined by the results themselves. This point was then bound to be one at which a clear distinction between the drugs, or a close similarity of them, had been established. The special form of analysis used makes due allowance for this characteristic of a sequential trial, which the simple form of calculation made by Dr. Trevan does not.

It would, of course, have been perfectly possible for us to have planned our trial in such a way as to have enabled us to establish, with calculable risks, the superiority of either calcium chloride or adrenaline. Unfortunately, as it now turns out, we did not plan to test the superiority of adrenaline. It will be understood that the trial would never have been begun had not the claims made for calcium chloride suggested that it was superior to more conventional remedies.

May we hasten to add that common sense has led us to the same opinion as Dr. Trevan's, that calcium chloride is

no good? We cannot, however, use a statistical argument to reinforce this opinion, and it was therefore not claimed as a result of the sequential trial. Nevertheless, there is no conflict between common sense and statistics here; we are all accustomed to act on common-sense opinions for which statistical justification is lacking. Common sense tells us the sun will rise to-morrow; although the odds that it will do so are probably high, they cannot be worked out.—We are, etc.,

G. S. KILPATRICK.
P. D. OLDHAM.

Penarth, Glam.

Antibiotic Sensitivity Spectra

SIR,—I have read with interest the article by Dr. E. P. Silberstein (*Journal*, January 15, p. 148) on the use of antibiotic spectra in the investigation of epidemics. It is, however, unfortunate that Dr. Silberstein used both aureomycin and terramycin for his series of tests, since the sensitivity of *Staphylococcus aureus* to these two antibiotics is almost always the same. The development of resistance to one of them automatically confers resistance to the other. Dr. Silberstein's method would have greater powers of differentiation if another antibiotic were used instead of terramycin.—I am etc.,

R. BLOWERS.

Middlesbrough.

Leukaemia and Ankylosing Spondylitis

SIR,—A conference was recently convened by the Medical Research Council to consider new ways of inquiring into the causes underlying the present rise in the death rate from leukaemia. At this conference a memorandum was presented drawing attention to an apparent association between leukaemia and ankylosing spondylitis. This information was based on a rather limited number of instances, and the members of the conference considered it very desirable that further evidence should be collected on this question. The possible association of leukaemia with ankylosing spondylitis is a curious one, but it may well be one that has been noted in individual patients by some of your readers.

Dr. W. M. Court-Brown, a member of the Council's External Scientific Staff, at present working at the Department of Medicine, The Postgraduate Medical School of London, Hammersmith Hospital, London, W.12, is collecting the relevant data on this subject. He would be very grateful if any of your readers who may have witnessed the occurrence of leukaemia in a patient suffering from ankylosing spondylitis would write to him.—I am, etc.,

H. HIMSWORTH.

London, S.W.1.

POINTS FROM LETTERS

Boxing Injuries

Dr. HERBERT SIMON (London, N.3) writes: I have read the conclusions of Drs. J. L. Blonstein and E. Clarke *re* amateur boxing (*Journal*, December 25, 1954, p. 1523) summing up that "amateur boxing is a useful and justifiable sport." In my opinion boxing is only justified as a weapon of defence. Only six deaths due to boxing among amateurs within the last seven years have occurred, so we are told. I have watched amateur boxing in reality and in good close-up shots on television. What a spectacle watching young men hammering at each other in a more or less skilful way! The principle in boxing is to knock the other fellow out by inflicting injuries to your opponent. The "sport" is designed to win by doing the most possible physical damage to your opponent and to hurt him as severely as possible. It is a sorry tale of the commentator we have to listen to of bleeding noses, wobbly knees, eyes blinded by blood, and the lovely pay-off right on the spot of the jaw. I cannot imagine that cranium, eyes, ears, and the solar plexus are created to be intentionally exposed to punishment which might sooner or later lead to physical or mental harm.

Obituary

GEORGE HALL, C.M.G., M.D., F.R.C.P.

Dr. George Hall, consulting physician to the Royal Victoria Infirmary, Newcastle-upon-Tyne, died on January 10, aged 75.

George Hall was the only son of M. Stokoe Hall, of Bishop Auckland. From Armstrong College, where he took the Durham degree of B.Sc. in 1899, he went on to study medicine at University College, London, where he was Bucknill Scholar and where he won a gold medal and exhibition in physiology at the intermediate M.B. examination in 1901. He also won the Cluff Prize in the same year. Graduating M.B. in 1903, he continued his brilliant student career by winning the Atkinson Morley Scholarship in 1904. He proceeded M.D. in 1905, took the M.R.C.P. in 1907, and was elected F.R.C.P. in 1927.

After qualification Hall held the posts of house-physician and house-surgeon at University College Hospital, and then was junior and senior house-physician at the National Hospital, Queen Square. Returning to Newcastle to take up consultant practice he was elected assistant physician and assistant pathologist at the Royal Victoria Infirmary in 1908. He also became assistant demonstrator of bacteriology at the College of Medicine at Durham University, which he was later to serve as registrar. One of the first papers he published in this *Journal* was on the bacteriology of keratitis. Hall was a Territorial, and during the first world war he served in the R.A.M.C. with the rank of major, being appointed C.M.G. in 1916.

In the years after the war Hall quickly established a large practice as a consulting physician, especially in neurology. He became a full physician on the staff of the Royal Victoria Infirmary in 1927, and lecturer in medicine at King's College. He had previously been lecturer in medical jurisprudence and in materia medica and pharmacy. Among other appointments he held were those of honorary physician at the Ingham Infirmary, South Shields, consulting neurologist to the Home for Crippled Children at Gosforth, and visiting physician to the Ministry of Pensions Hospital, Dunstan Hill. He was also a Fellow of University College, London, a J.P. for Newcastle, and medical visitor for the Durham county magistrates. During the Annual Meeting of the B.M.A. at Glasgow in 1922 he was vice-president of the Section of Medicine; and he was chairman of the Newcastle-upon-Tyne Division of the B.M.A. in 1931–2.

Dr. Hall married Margaret, eldest daughter of Nicholas Mein, of Newcastle. They had one son and one daughter.

J. H. B. writes: I would like to pay a tribute to my old friend Dr. George Hall, who was a very lovable man. He was good-looking, modest and quiet, and a good conversationalist. He had a very kind and friendly disposition and was always ready to help anyone who came to him for advice. He was popular with colleagues, students, and nurses. He was one of the old school and most conscientious in his hospital attendances. In spite of a very busy professional life he found time to indulge in some hobbies. He was fond of music, golf, and cricket, thoroughly enjoying taking part in the annual cricket matches between the staff of the infirmary and the students. For the past few years, after the death of his wife and the departure south of his son and daughter, Dr. Hall was a rather lonely man.

He loved his friends to call in and have a chat by the fireside. About two years ago he had a serious illness from which he never fully recovered. He will be greatly missed in Newcastle. Our sympathy goes out to his son and daughter.

H. K. WALLER, M.B., F.R.C.O.G.

After a long and distressing illness Dr. H. K. Waller died on January 13, aged 73, at the home of his adopted daughter in Wadhurst. For most of his life he was a general practitioner, but his infant welfare work at the British Hospital for Mothers and Babies, Woolwich, and elsewhere brought him well-deserved renown.

Harold Kirk Waller was born in 1881, the only son of the late Rev. Horace Waller, who was in his early days a companion of David Livingstone in some of his later journeys and after whom Waller's gazelle (*Lithocranius walleri*) was named. Horace Waller came much into contact with Sir John Kirk, British Agent at Zanzibar for many years, the adviser and helper of all the mid-Victorian East African explorers, and for this reason his son was given the name Kirk. Harold Waller was educated at Charterhouse School and at Trinity College, Cambridge: thence he went on to the medical school of University College Hospital, qualified M.R.C.S., L.R.C.P. in 1909, and graduated M.B. in 1911. He was house-physician at his own hospital and at the Shadwell Children's Hospital as well as resident medical officer at Queen Charlotte's Hospital. After service in the R.A.M.C. during the 1914–18 war he was in general practice in Chelsea, and there developed a special interest in infant feeding and infant welfare; but he always insisted that he was primarily a general practitioner, that mother and baby should be treated as a unit, and that it is the family doctor and nurse, provided they are adequately trained, who can best manage and supervise the nursing couple. In 1914 he had been appointed superintendent medical officer at St. Katharine's College, Poplar. This was a child health centre, started with funds provided by the Chapter of London, at which there were infant welfare, dental, and sunlight clinics, as well as eight to ten cots for in-patients. His appointment there continued until the inception of the National Health Service. He always had a warm affection for those who lived in the East End, and during the second world war was placed in charge of the children's ward at the Poplar Hospital, at the same time being appointed physician to the nursing staff.

Waller's association with the British Hospital for Mothers and Babies at Woolwich began in 1913 and continued until 1950. During the years since his retirement he still kept in touch with the hospital, and in the latter part of his illness, when travelling from Tunbridge Wells was a physical strain, made every effort to visit the hospital, which had become a Mecca for all those interested in the subject of lactation and the care of the newborn. He was elected an honorary F.R.C.O.G. in 1953, the second paediatrician in this country to be so honoured, and in 1951 received the Dawson Williams Prize from the British Medical Association in recognition of his original contributions to the clinical study of lactation and infant feeding.

The Chelsea Babies' Club was another institution to which Waller was devoted. He was its first medical adviser when it was started in 1927 with Lady Piercy as its chairman. Here he instituted classes for expectant mothers, instruction on infant care, breast-feeding, suitable clothing, the importance of open air, nursery planning, and so on. He was a shrewd observer and an excellent and discriminating writer. In *Clinical Studies in Lactation* (published in 1938) he described all that he had learnt about lactation—for example, the draught reflex which governs the outflow of milk from the breast, the reasons for early failure of breast-feeding and the re-establishment of lactation. His observations were made at the bedside, in the out-patient clinic, or in the patient's home without resort to esoteric laboratory procedures. His wish was that there should be two rooms at the Woolwich Hospital for "after-care" of mothers and babies. The building of this annexe with money collected from the nursing staff, doctors, and patients is almost complete—an appropriate memorial to him. He had been a widower for a few years and leaves no issue.

Sir EARDLEY HOLLAND writes: It was characteristic of Waller to head his last piece of writing with a quotation from his admired teacher, Wilfred Trotter: "The scientific world is a democracy of facts where the inhabitants are of equal standing and there is no table of precedence. Indeed, the progress of knowledge has been in great part the discovery of the importance of small things and the exaltation of the insignificant." Waller's interest in obstetrics no doubt began as R.M.O. at Queen Charlotte's, and when he went into practice in Chelsea he became in due course what would now be called a general-practitioner obstetrician. The advantage this sort of practitioner has over the obstetric consultant accoucheur is that he has the continuous care of the baby from birth onwards and the welding of the mother–baby unit. He soon became deeply interested in breast-feeding (his "last phase of the reproductive cycle"), the clinical study of which was strangely neglected, and the want of success of which, as conventionally managed, was deplorable. Even a recent survey showed that lactation failed within eight weeks of delivery in nearly one-half of nursing mothers. When Waller began practice the position can have been no better; but instead of passively accepting it he began to dedicate himself to a lifelong clinical study. In such regard did he hold baby welfare that he founded a private infant welfare centre, the Chelsea Babies' Club. His ideas soon became known to leading London midwives and welfare workers, for here was a man who seemed likely to solve one of their most hopeless problems. His work soon gathered impetus: in 1914 he became medical superintendent to the earliest public welfare clinic in London (The Royal College of St. Katharine) and physician to the Home for Mothers and Babies, Woolwich, which led on to his later appointments as paediatrician to Poplar Hospital and to the British Hospital for Mothers and Babies, Woolwich. The secrets of lactation were revealed to Waller by patient and persistent observation, through the years, of babies at the breast and by careful recording, whereby he was able to form conclusions that were convincing and to recommend simple devices that were at once adopted. Groups of devoted midwives and co-workers helped, above all at the Woolwich Hospital, which became the recognized centre of his teaching. Midwife teachers and sisters, and even young obstetricians, were sent there to learn, not only from British centres, but from many parts of the world, and he visited many centres by invitation to show his methods.

Medicine was a vocation to Waller, and from first to last he was a dedicated man. Throughout his life he inspired a feeling of affection in all who came in contact with him. Such an unusual combination of gifts—charm, gentleness, unselfishness, quick sympathy, and devotion—captured all hearts. As a doctor he was great, but he was greater still as a human being. So lived one who was content with the discovery of small things and the exaltation of the

insignificant. But was the bringing of so much happiness and comfort to so many mothers and babies such a very small and insignificant thing?

Mr. T. TWISTINGTON HIGGINS writes: Harold K. Waller was one of the outstanding doctors of my time. Pioneer in the natural feeding and welfare of infants—who can compute the debt owed to him, or yet to be incurred, by babies and their mothers? All who were fortunate to count at need upon his steadfast care and skill in their own homes can never forget this prince of family physicians. To those privileged to enjoy his friendship there will ever remain the inspiring memory of his commanding debonair figure, gentle courtesy, and infinite kindliness of heart. His alert mind with its own gifts of insight, quiet humour, and modesty made him a delightful and stimulating companion, whether in a teeming noisy Poplar street, in the charm of his Chelsea house, or among the flowers and peace of his beloved country garden. At Charterhouse he had played football in distinguished company, with some whose names are now a legend—G. O. Smith, Oakley, Cobbold—and much of the boy always remained to temper the dignified distinction of the man. Harold Waller made his world a richer place for many, and those who knew him best are sadly the poorer for his passing.

Dr. DAVID MORRIS writes: Dr. Waller will be most remembered by his pioneer work on breast-feeding. But we who knew him personally know intimately the remarkable quality that lay behind his scientific contribution. Whether on ward round or in lecture-room, at consultation or in social gathering, his special ability was always to establish a warmth of human relationship. His work was much more than a mere method or technique of antenatal preparations or post-natal management of breast-feeding; it was the application of a rich experience and judgment to individual human beings. He was a remarkable example of the old school who learned and taught on the individual case rather than by statistical analysis. Typical of his mode of working was his last series of clinical observations, carried out after his retirement, on a small group of individual nursing mothers whom he visited in their homes; his accurate observations added much information to the natural history of breast-feeding. He was held in high esteem by all who knew him, exemplified in the building of the "Dr. Waller Rooms" at the British Hospital for Mothers and Babies, Woolwich, from a voluntary fund launched after his retirement.

J. D. WHITE, O.B.E., M.B., D.M.R.E.

Dr. J. D. White, director of the radiological department at the Hammersmith Hospital and senior lecturer in radiology at the Postgraduate Medical School, died in London on January 21 at the age of 59. He had suffered for years from the effects of working with radium during his early years as a radiologist.

John Duncan White graduated M.B., Ch.B. at Edinburgh University in 1917 and held the post of resident surgeon at the Royal Infirmary. During the closing stages of the first world war he was a captain in the R.A.M.C. On his return to civilian life he determined on a career in radiology, and served his early years under the guidance of his revered chief, the late Dr. Stanley Melville. In 1921 he took the D.M.R.E. of Cambridge, having held the post of clinical assistant in the *x*-ray department of King's College Hospital. After working for some years as assistant radiologist at St. George's Hospital and at the Brompton Hospital, and as radiologist at the Hospital of St. John and St. Elizabeth and the Hospital for Tropical Diseases, he was appointed in 1932 as radiologist at the Royal Infirmary, Edinburgh, and also as lecturer in radiology at the University. Within two years, however, he was back in London as director of the radiological department at Hammersmith Hospital, where he had been working for over 21 years at the time of his death.

Indefatigable in the efforts he made for the good of his specialty, Dr. White served on the councils of many organizations concerned with radiology. For many years he was a member of the council of the British Institute of Radiology, being elected its president in 1934, and serving as honorary treasurer from 1939 to 1945. In 1936 he was vice-president of the Section of Radiology at the Annual Meeting of the B.M.A. in Oxford, and he was a member of the Consultants Board and of the Radiologists Group Committee of the B.M.A. for a number of years. Much of the credit for the high standard of radiography set by the Society of Radiographers must be given to him, for he had been one of its critical and constructive advisers throughout. During the last war he served again in the R.A.M.C., attaining the rank of temporary major. He was appointed O.B.E. in 1953.

Dr. JAMES F. BRAILSFORD writes: With the passing of John Duncan White another doctor joins the roll of *x*-ray martyrs, and British Medicine has lost one of its greatest exponents in radiology, radiologists a loyal and courageous colleague, and radiographers their best adviser and friend. White rightfully looked with disfavour on that modern appropriation of radiological services in experimental medicine which was activated merely by desire to satisfy the curiosity of the investigator—the risks to the patient being ignored. Though he taught and showed the highest standards of radiology to his students, radiographers, and radiologists, he did not consider the production of a spectacular radiograph his goal, but merely a means to help the patient. The control of a big department, in which the chief so scrupulously and intimately inspected all the work and advised with firm humanitarian principles on all of its most important investigations, may not produce universal approval, but such behaviour did endear all to him who honoured good work. His integrity, enthusiasm, and desire to render to his patients the utmost service radiology could give unfortunately resulted in severe radiation burns which necessitated several amputations. These ominous signs, though associated with much pain, were borne silently with great fortitude. He made light of his sufferings and kept his friends free from any painful reference to them, and by his quiet and courageous facing of an early death put the hall-mark on the enduring love and affection of his devoted wife, Claire, to whom we extend our deepest sympathy. Truly can it be said of him as of other *x*-ray martyrs:

"But in our hearts they rest
Not less revered than blest
Than those their brothers who in fighting fell,
Nor shall our children hear their names pronounced less dear
When England's roll of gallant deeds we tell."

A colleague writes: Duncan White was a most gallant fellow. No one who knew him and saw him hobbling about, overcoming his physical disabilities and doing his work day after day under circumstances which were often distasteful, would deny him this merit. Thrust into radiology in the urgent days of the 1914–18 war, with little experience and no knowledge of its perils, he sustained damage which ultimately caused his death. Knowing his inevitable end, he faced the long-drawn-out period of waiting with a courage which shamed those who occasionally visited him in those latter days. We may mutter *Ave atque vale* and feel that we have paid some tribute to a man of great heart and cool courage, but until and unless we face the same ordeal we can never evaluate the gallantry which D. W. brought to meet it.

R. J. WILLAN, C.B.E., M.V.O., M.S., F.R.C.S.

Sir GORDON GORDON-TAYLOR writes: Perhaps I may be granted a few lines to add a personal tribute to the late Professor R. J. Willan—"R.J." to his friends—whose obituary notice appeared in the columns of last week's *British Medical Journal*. I have been deeply indebted to Willan on at least two occasions: first during the earlier part of the 1939–45 war when he was consulting surgeon in the north-east region of England and I was busying myself with the collection of abdominal casualties due to air-raid attacks on Britain; and, secondly, I cannot forget the generous help which he afforded me in the preparation of the Rutherford Morison Centenary Lecture with which I was honoured in 1953. Rutherford Morison remained his hero till the very end of life, and, although I should not dare to decide whether Charles Saint, of Capetown, or Willan was Morison's "beloved disciple," the duration of "R.J.'s" friendship and loyalty was inevitably the longer. The numerous letters from Morison to Willan which the latter had preserved and which he kindly permitted me to read showed that the affection was not one-sided: it was to Willan and Hamilton Drummond, assisted by Gordon Irwin, that Morison turned for surgical help in 1923, and their surgery prolonged his life for 15 years. And I have also in my possession a copy of the pathetic letter written in 1938 by Morison, who when 85 years of age was faced with a serious surgical ordeal, dropping "the old pilot": "the old pilot" paid his last tribute to his chief a few days later in the chapel of the R.V.I., the scene of their long comradeship.

Perhaps Willan was more proud of his association with the Royal Navy than almost anything in his long life: many are the surgeons of the Royal Navy who are eternally grateful to him for appointments and surgical registrarships in the R.V.I., Newcastle. The medical department of the Admiralty was not unmindful of this loyal help to the Senior Service, and, when opportunity suddenly offered during the last world war, there was not a moment of delay, and Willan, to his unfeigned delight, donned the uniform of a surgeon rear-admiral. He may not have been one of the world's master surgeons; he may not have even been an eloquent and forceful teacher; but he had a genius for friendship, and, as he wrote of Rutherford Morison, he also overcame obstacles by sheer force of character, merit, and good temper; and to so many, as to his old chief, he will always remain "my old and loyal friend," utterly incapable of any mean or ungenerous action.

J. B. MILLER, M.D., LL.D., D.P.H.

The obituary notice of Dr. J. B. Miller was published in last week's *Journal* (p. 232).

We are indebted to Dr. JOHN LAMBIE for the following appreciation: My first contact with "J. B." was when we were students. He was always a studious chap and paid great attention to his work, but, as the fruits of that are well known and recorded, I would like to make some mention of his other interests.

His earlier interest, other than medicine, was centred in the wild flowers of Scotland, consequently botany in his first year was of great interest: he attended the excursions and collected a herbarium which was the envy of many. Later in life he became a great gardener and never was happier than when pottering in his garden and hot-house. Last year a severe storm reduced his hot-house to the ground and he lost many of his cherished plants, but his philosophy helped him through and he continued his interests in his outdoor plants and shrubs, knowing them as friends and full of knowledge of their special characteristics.

When a student he played Rugby and was a member of the medical team when the interfaculty matches took place. He enjoyed the rough and tumble, and his interest in Rugby remained all his life. He was a keen supporter of his old school F.P. team (Glasgow High School) and attended the matches faithfully on Saturday afternoons religiously wearing his "old school tie." Murrayfield also engaged his attention when the internationals were on. He was also very keen on golf. Apart from playing on his own course, he was for many years a member of the Glasgow Medical Golf Club and attended steadily during that time. Curling in the Glasgow rink claimed his attention when it was possible, and he was very keen, not only for the game, but because he also met his friends. He liked company and was a member of several medical dining clubs. Not only did he enjoy his meal, but his ability as an after-dinner speaker was greatly in demand. He replied to many different toasts in his usual pawky and subtle manner, not forgetting to mix grey matter with humour and often introducing a Latin quotation. As a man he was a kindly, erudite family doctor who was not only sympathetic with his patients as regards their health but also was a great help in private problems. As such, his passing, while being a great loss to his patients and the community, will be keenly felt by his large circle of friends.

Dr. J. W. WILSON, who had been in general practice at Stirling since 1925, died in Edinburgh on December 31, 1954, at the age of 59. John Wight Wilson was born on April 4, 1895, and was educated at Glasgow University. As an undergraduate he left his medical studies in 1914 to serve with the Highland Light Infantry in the first world war, and saw active service as a combatant officer in France and Mesopotamia. He returned to Glasgow after the war, and, after graduating M.B., Ch.B. in 1922, held house appointments for the next eighteen months at the Western Infirmary and the Royal Maternity Hospital. He then spent a year as an assistant at Cumnock, Ayrshire, before moving in 1925 to Stirling, where he was successively assistant, partner, and, later, senior partner in a general practice. In 1936–7 he was president of the Stirling Branch of the British Medical Association. In 1926 he married Miss Catherine A. S. Blair, who graduated M.B., Ch.B. with him in 1922.

W. B. G. A. writes: For 30 years Dr. Wilson's work in Stirling was characterized by an integrity and a thoroughness which were based on a very lively Christian faith. In addition to the work of a large practice he made time and found the strength to give assistance to the Boys' Brigade, and, when need arose, to the Home Guard and the Army Cadet Force. In all his work, and to his family, he gave himself to the utmost, never counting the cost. His spirit lives on, and in our memory of him we have a very precious heritage. He leaves a widow, a son in the Royal Navy, and four daughters, all of whom are in work having some connexion with medicine.

Dr. G. A. WILKES, a well-known Birmingham practitioner, died at his home on January 8 at the age of 84. George Arthur Wilkes was born on July 26, 1870, and was educated at Queen's College, Birmingham, and at Leeds, qualifying M.R.C.S., L.R.C.P. in 1892. Two years later he obtained the D.P.H. of the English Royal Colleges. In 1901 his name appeared in the first list of recipients of the M.B., B.Ch. degrees granted by the newly founded Birmingham University, and he was awarded an Ingleby Scholarship and the senior medical prize. In the following year he proceeded to the M.D. He held the appointment of resident surgical dresser at Queen's Hospital, Birmingham, and was in practice at Bradford, Yorkshire, for a short time before returning to Birmingham some three years before the turn of the century. At Trinity Road, Aston, he quickly established himself as an outstandingly good doctor, and later he added to his work by becoming medical officer and public vaccinator for Aston Manor, medical officer to the Post Office and to the Birmingham Fire Brigade, and police surgeon. Becoming increasingly troubled by deafness, he retired from practice about ten years ago. He leaves a widow, a son who is also in general practice, and a daughter.

We are indebted to Dr. H. GUY DAIN for the following appreciation: With the passing of George Arthur Wilkes, Birmingham loses one of her notable and oldest doctors.

A product of the Birmingham Medical School in the days before the University had come into being, he started practice with the Conjoint qualification in 1892. Although he soon attracted a good general practice, he had the energy to take a D.P.H. and, when they became available, to work for and to take the M.B., B.Ch. degrees of Birmingham and, later, the M.D. in 1902. He took a great interest in medical affairs and in the British Medical Association, holding office as chairman of the Birmingham Central Division from 1916 to 1918. In 1923–4 he was president of the Birmingham Branch. As a member of the local medical and panel committee for many years, he became chairman in the early years of National Health Insurance. He showed the same interest in the academic side of medicine and was president of the Midland Medical Society. He was a sound clinician, and his opinions on cases, or on medico-political problems, were always well considered and showed good judgment. These opinions could be forcibly expressed. He once said that the smallest object that a person should be allowed to put in his ear was his elbow. Perhaps it is hardly necessary to say that he could hold his opinions very firmly in debate. Off duty he enjoyed, until late years, his game of golf, and a game of bridge was always an attraction. During the last war, after an operation which was followed by very dangerous conditions, he made a good recovery and came up smiling to enjoy his retirement.

Mr. L. A. KEY died suddenly at his home at Ascot on January 9 at the age of 55. Leonard Arthur Key was born at Norwich on December 15, 1899. He received his early education at the local grammar school and then served as a midshipman in the R.N.V.R. in the first world war. He went to Cambridge in 1918 and completed his medical studies at St. Thomas's Hospital, qualifying with the London conjoint diploma in 1925. Following a year's appointment as house-surgeon at the East Surrey Hospital, Redhill, he entered general practice at East Grinstead. During 1932 indifferent health, undoubtedly caused by the fact that he never spared himself in the interest of his patients, forced him to take up a less strenuous branch of medicine, and following a year with the late Sir Henry Gauvain at the Lord Mayor Treloar Orthopaedic Hospital, Alton, he developed a great interest in bone and joint tuberculosis and entered the L.C.C. health service in 1934. While in this service he became deputy medical superintendent of Queen Mary's Hospital for Children, Carshalton, and in 1947 was appointed medical superintendent of Heatherwood Orthopaedic Hospital, Ascot. During this time he wrote several papers on bone and joint tuberculosis affecting the hip and knee.

The following appreciation has been jointly written by several colleagues of Mr. Key: At Heatherwood Mr. Key's wide knowledge of medical administration, combined with a sound and broadly based judgment, enabled him to guide the development of the hospital from the treatment only of long-term bone and joint tuberculosis to its present function as a regional orthopaedic centre. The original conception was largely his own, and he worked quietly, tactfully, and unswervingly towards that end. He was a member of the regional orthopaedic committee and was able extensively to influence both policy and detail, always modestly but with clear-sighted and lucid argument. Modern advances in prevention and treatment of tuberculosis reduced his chosen field of work, and, moving with the times and in company with his hospital, he gained the F.R.C.S.Ed. in 1949, and extended his clinical activities into the general range of orthopaedic surgery. He was particularly interested in the problems arising from poliomyelitis, and many young people will walk through life more effectively for having passed through his patient hands. His approach to an orthopaedic problem combined the outlook of a research worker with that of a clinician. Careful dissection in the anatomy school, discussion with colleagues, examination of many cases, critical perusal of current literature, all led to a logically reasoned opinion and an operation planned in detail beforehand and executed with thought and skill. He gave great encouragement to his registrars and had a way of teaching that was most discreet. No young man could work with him without thereby improving his surgery or realizing that he had a friend with his welfare at heart.

Dr. FRANK BODMAN writes: In your obituary of the late Dr. Elizabeth Casson (*Journal*, January 1, p. 48) no mention was made, among her manifold medical interests, of her membership of the Faculty of Homoeopathy. Dr. Casson joined the British Homoeopathy Society in 1927 and applied the principles of homoeopathic medicine in the medical treatment of her psychiatric patients with considerable success. As late as last summer she made a valuable contribution to a discussion at the British Homoeopathic Congress on homoeopathy and geriatrics, based on many years' clinical experience. She had been a fellow of the Faculty for some years.

Medico-Legal

DEATH FOLLOWING OBLIVON AND NEMBUTAL

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

On November 26, 1954, Frank Ronald Preston, aged 23, died after taking about 20 "nembutal" tablets and about 9 "oblivon" capsules.

The post-mortem examination was carried out on the same day by Dr. W. K. Taylor, who found gross oedema and venous congestion of the lungs. A striking feature of the abdomen was the presence of intense venous congestion, causing marked distension of the veins. Dr. Taylor gave the cause of death as acute pulmonary oedema and cardiac failure consistent with narcotic poisoning, pulmonary oedema being sometimes caused by the barbiturates.

The deceased's stomach and duodenal contents, small intestine and caecum, liver, kidneys, and spleen, together with a portion of the lung and a sample of the urine, were sent to the Metropolitan Police Laboratory, where they were examined by the director, Mr. L. C. Nickolls, F.R.I.C. He identified the presence of methylpentynol in quantity equivalent to about 9 oblivon capsules, and about 7½ gr. of a barbiturate the identity of which he could not completely confirm because of the small quantity present, but which he thought to be nembutal.

At the inquest held at Godalming on December 13, 1954, in answer to the coroner, Dr. J. M. Robertson, Mr. Nickolls said in evidence that, while the amounts of oblivon and barbiturate present were not in themselves sufficient to cause death, it had been shown that the two were synergic in action and that similar amounts together had proved fatal. Oblivon was an acetylene alcohol which it was not desirable to take together with barbiturate. The result was similar to that of alcohol and barbiturates taken together.

An open verdict was recorded, the coroner finding that there was insufficient evidence to show why or under what circumstances the oblivon and nembutal had been taken.

SURGEON SUES DOCTOR FOR FEES

Judgment by consent for £183 15s. and costs was entered for a gynaecological and obstetric surgeon in the Queen's Bench Division on January 20 when he sued a doctor for fees alleged to be owing in respect of professional services to the doctor's wife. She died on November 30, 1952, aged 53. The surgeon claimed £265 2s. 6d., which he alleged was owing in respect of professional services up to November, 1952. The husband was sued as executor of his wife's estate. The three people had first met in Berlin before coming to Britain.

The defence was that it was a medical custom for doctors not to charge each other for professional services. There

was said to be an agreement between the surgeon and the doctor's wife that the treatment should be free; and the defence claimed that the fees were excessive and that the surgical operation on the defendant's wife was performed without the defendant's authority and unskilfully. The surgeon denied these allegations.

The doctor's wife consulted the surgeon in November, 1950, and paid him £9 17s. 4d. She never paid anything for subsequent treatment, and the plaintiff did not ask for payment because he did not wish to embarrass her financially, preferring to wait until she was better placed. She was separated from her husband. The surgeon said in evidence that on November 18, 1952, the patient telephoned him, and on examining her he discovered a growth. He decided that an operation was necessary. She refused to go into hospital and said she would prefer to go into a nursing-home under his care, despite the extra expense. She refused to have a second opinion. The surgeon said that when he saw the doctor on December 3, 1952, he did not complain about the treatment nor did he say it was wrong to operate. Later the doctor told him he was surprised at the amount of the fees.

Cross-examined, the surgeon agreed that there was a wide practice among medical men not to charge each other for professional services, but it was not a binding practice. During the cross-examination the defendant received advice from his counsel, and after an adjournment the surgeon agreed to accept judgment for £183 15s. and costs against the doctor. The doctor withdrew unreservedly the allegations of negligence and want of skill, and was satisfied that plaintiff did everything that could be done.

The judge said he could well understand that the surgeon had to fight the case and bring these proceedings. He also thought the doctor had acted wisely, having heard what the surgeon had to say, in recognizing that there was no foundation for the allegations.

Vital Statistics

Influenza

Influenza deaths in the 160 great towns of England and Wales increased from 102 in the preceding week to 134 in the week ending January 15. All regions showed increases except the southern, south-western, and Wales, the figures for which remained practically the same. 86.6% of the deaths were of people aged 55 and over and 70% of people aged 65 and over. There is little change in the situation. Many of the outbreaks are due to virus-B influenza, though isolated cases of virus-A infection have been reported in the midlands and Wales. On the whole the prevalence of influenza seems to be waning in many areas and spread elsewhere to be slow.

Record Low Infant Mortality

Provisional figures for England and Wales in 1954 show that the infant mortality rate for the year was the lowest ever recorded. There were 17,098 deaths of children under 1 year of age, giving a rate of 25.5 per thousand related live births. This rate compares with 26.8 in 1953, 27.6 in 1952, 29.7 in 1951, and 52.8 in 1938. Infant deaths in the December quarter numbered 4,067, giving a rate of 25.3. This also is a record figure for a fourth quarter, the previous lowest rate for that quarter being 26.2 in 1953. The total deaths numbered 501,878, giving a rate of 11.3 per thousand population, compared with rates of 11.4 in 1953 and 11.3 in 1952.

There were 16,130 stillbirths, giving a rate of 24.0 per thousand total live and stillbirths. This was higher than the rates for 1953 and 1952 of 22.5 and 22.7 respectively.—Registrar-General's *Weekly Return*, No. 2 (H.M.S.O.).

Week Ending January 15

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 465, whooping-cough 1,769, diphtheria 14, measles 7,800, acute pneumonia 1,121, acute poliomyelitis 22, dysentery 402, paratyphoid fever 2, and typhoid fever 3.

Infectious Diseases

The chief feature of the returns of the notifications of infectious diseases in England and Wales during the week ending January 8 was the large increase of 1,065 for measles, from 8,320 to 9,385. The only other variations of note were a fall of 244 for scarlet fever, from 707 to 463, and a rise of 69 for whooping-cough, from 1,722 to 1,791.

The largest rises in the incidence of measles were 331 in Lancashire, from 2,510 to 2,841, 269 in Yorkshire West Riding, from 703 to 972, 127 in Nottinghamshire, from 215 to 342, and 117 in Cheshire, from 396 to 513, and the largest fall was 54 in Warwickshire, from 327 to 273. No large fluctuations were recorded in the local trends of whooping-cough. The largest decrease in the number of notifications of scarlet fever was 36 in Yorkshire West Riding, from 75 to 39. 13 cases of diphtheria were notified, being the same number as in the preceding week.

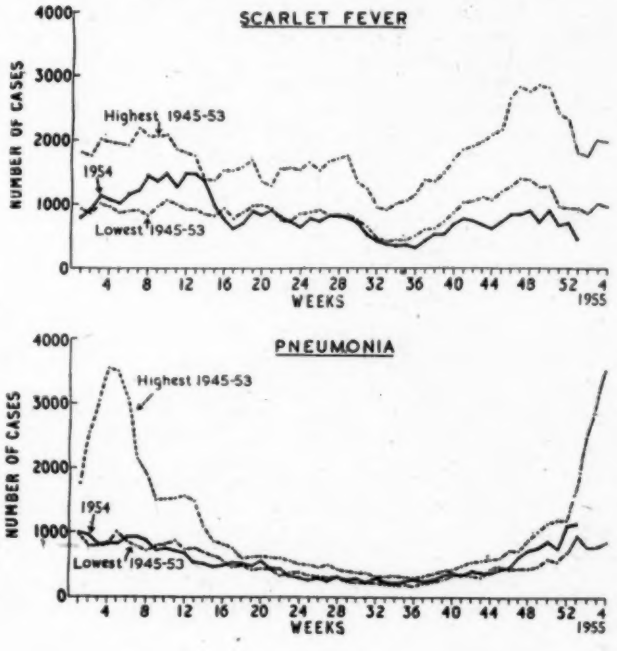
25 cases of acute poliomyelitis were notified; these were 1 more for paralytic and 1 more for non-paralytic cases than in the preceding week. The largest returns were Lancashire 6 (Liverpool C.B. 4, Manchester C.B. 2) and London 4.

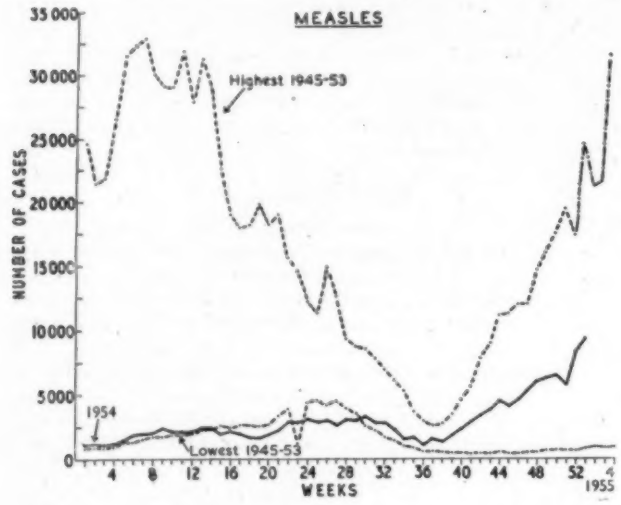
346 cases of dysentery were notified, being an increase of 6 on the total of the preceding week. The largest centres of infection were Norfolk 60 (Norwich C.B. 35), Lancashire 48 (Oldham C.B. 14), Yorkshire West Riding 43 (Sheffield C.B. 18, Leeds C.B. 11), Cheshire 32 (Marple U.D. 10), Lincolnshire 26 (Scunthorpe M.B. 24), and London 20.

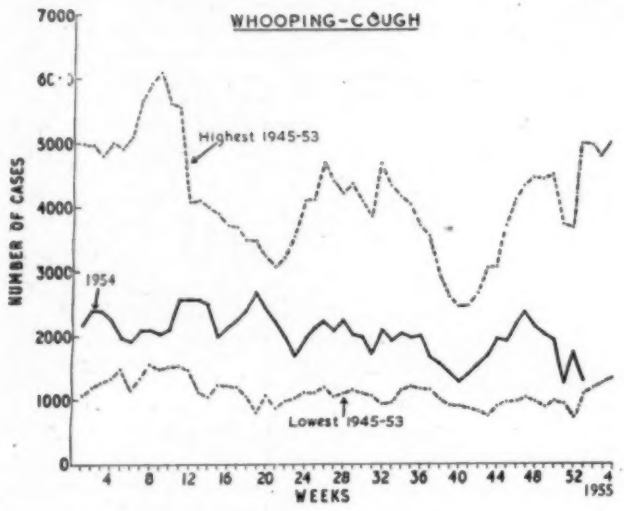
102 deaths were attributed to influenza in the great towns, 20 more than in the preceding week. The largest return was 7 from Exeter C.B.

Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported in each week during the nine years 1945–53 are shown thus - - - - - -, the figures for 1954 thus ————. Except for the curves showing notifications in 1954-5, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.







Infectious Diseases in 1954

The incidence of infectious diseases in England and Wales during 1954 was low, and for most of the diseases the number of notifications was the lowest in recent years. The chief exception was dysentery. The number of notifications of this disease was a record, being 15% above the previous high level of 1951. The number of notifications of typhoid and paratyphoid fever, although low, was in excess of the number in 1950 and 1953. The number of notifications of diphtheria was the lowest ever recorded, and was only 1/84 of the number in 1938. The notifications for the past five years were as follows:

England and Wales	1950	1951	1952	1953	1954
Scarlet fever ..	66,852	49,431	69,091	61,578	43,388
Whooping-cough	157,714	169,394	116,311	157,772	105,628
Measles	366,116	615,332	400,838	534,020	147,076
Acute poliomyelitis	8,699	3,093	4,504	5,251	2,388
Diphtheria ..	2,858	1,969	1,473	1,022	778
Meningococcal infections ..	1,745	1,956	1,687	1,783	1,569
Dysentery ..	18,135	29,488	15,665	19,540	33,976
Acute pneumonia	30,714	43,181	31,403	37,058	26,750
Enteric fevers ..	654	1,438	1,190	569	751
Great towns: Influenza deaths			996	3,381	947

Smallpox in France

Up to January 14, 44 cases of smallpox had been reported in the Vannes area, including 5 deaths (6 according to press reports).

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **January 8** (No. 1) and corresponding week 1954:

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1955 Eng. & Wales	1955 Lond.	1955 Scot.	1955 N. Ire.	1955 Eire	1954 Eng. & Wales	1954 Lond.	1954 Scot.	1954 N. Ire.	1954 Eire
Diphtheria	13	2	1	0		21	1	10	1	
Dysentery	346	20	192	22	8	322	75	151	1	21
Encephalitis, acute ..	1	0	0	0		2	0	0	0	
Enteric fever:										
Typhoid	4	0	0	0	1	2	0	0	2	
Paratyphoid ..	4	2	1	0	0	1	0	2	0	
Food-poisoning ..	124	37		1		141	10		0	
Infective enteritis or diarrhoea under 2 years		3		17	12				9	24
Measles*	9,385	1004	228	434	101	1,134	13	24	24	120
Meningococcal infection	17	2	24	0	2	29	3	19	0	
Ophthalmia neonatorum	28	2	5	0		32	1	4	0	
Pneumonia† ..	1,135	75	330	1	1	994	51	454	11	
Poliomyelitis, acute:										
Paralytic ..	22	3	4	1	2	35	4	7	3	4
Non-paralytic ..	3	1				9	2			
Puerperal fever§ ..	245	48	13	0		212	29	12	1	1
Scarlet fever ..	463	47	113	41	35	792	47	198	31	27
Tuberculosis:										
Respiratory ..	642	65	89	9		608	71	113	18	
Non-respiratory ..	107	8	19	1		92	8	11	4	
Whooping-cough ..	1,791	120	140	37	22	2,156	83	347	33	62

DEATHS in Great Towns	1955 Eng. & Wales	1955 Lond.	1955 Scot.	1955 N. Ire.	1955 Eire	1954 Eng. & Wales	1954 Lond.	1954 Scot.	1954 N. Ire.	1954 Eire
Diphtheria	0	0	0		0	0	0	0	0	0
Dysentery	1	0		0		0	0		0	
Encephalitis, acute ..		0			0		0			0
Enteric fever ..	0	0	0			0	0	0	0	
Infective enteritis or diarrhoea under 2 years	12	0	1	1	2	10	2	1	0	3
Influenza	102	7	5		1	42	2	4	0	0
Measles	0	0	0	0	0		0	0	0	0
Meningococcal infection	0	0	0				1	0		
Pneumonia ..	417	70	36	10	5	385	56	36	10	5
Poliomyelitis, acute	4	1			0	1	0	1		0
Scarlet fever ..	0	0	0	1			0	0	0	0
Tuberculosis:										
Respiratory ..	91	14	12	2	4	129	15	20	5	7
Non-respiratory ..		0	1	0	1		1	1	1	2
Whooping-cough ..	2	0	0	1	0	1	0	1	0	0
Deaths 0–1 year ..	240	41	38	9	15	270	26	54	11	11
Deaths (excluding stillbirths) ..	6,827	978	775	128	196	6,811	909	774	123	175
LIVE BIRTHS ..	7,645	1087	1043	224	319	7,821	1115	1198	256	451
STILLBIRTHS ..	219	11	31			201	20	31		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Medical News

Princess Louise Children's Hospital.—Medical staff of the Princess Louise (Kensington) Hospital for Children are expected to oppose a recommendation to the board of governors of St. Mary's Hospital that the Princess Louise Hospital should ultimately be converted entirely to adult use. This recommendation is made by a committee appointed by the board of governors to review the services provided by the St. Mary's Teaching Hospital Group. The committee argues that the proportion of children's to adult beds in the group is too high, especially in view of the long waiting-list for adult admissions. The adult waiting-list stands at over 1,600, while that for children was 235 at the beginning of this month, and of these 159 were E.N.T. cases. This disproportion, according to the committee, reflects the notable downward trend throughout the whole country in the demand for hospital accommodation for sick children. There is also the successful start of the experiment in the home care of seriously ill children to be taken into account. The committee suggests as a beginning the temporary conversion of two wards at Princess Louise Hospital to make room for maternity cases from St. Mary's while alterations are being made there to accommodate the new obstetric unit. Earlier the committee recommended the conversion of the Paddington Green Children's Hospital, but now think it simpler to convert the Princess Louise Hospital.

Rockefeller and Dorothy Temple Cross Fellowships.—The Medical Research Council invites applications for the following travelling research fellowships for the academic year 1955–6. Full details and application forms, which must be returned not later than March 15, are obtainable from the Council's headquarters office at 38, Old Queen Street, London, S.W.1.

Rockefeller Travelling Fellowships in Medicine.—These fellowships are intended for graduates resident in Britain with some previous research training in either clinical medicine or one of the basic medical sciences who are likely to benefit from a period of work at a centre in the United States or elsewhere abroad before taking up teaching or research positions in the United Kingdom. Maintenance allowances for fellowships in the United States are normally at the rate of $2,700 per annum for a single fellow and $3,900 per annum for a married fellow. Provision is also made for travelling and other approved expenses.

Dorothy Temple Cross Research Fellowships in Tuberculosis.—These fellowships are intended to provide special opportunities for study or research to suitably qualified British subjects "intending to devote themselves to the advancement by teaching or research of curative or preventive treatment of tuberculosis in all or any of its forms." Maintenance allowances are at the rate of £650 per annum for a single fellow and £900 per annum for a married fellow, subject to adjustment for fellows working at centres outside the sterling area. Provision is also made for travelling and other approved expenses.

Lady Tata Memorial Trust.—The trustees invite applications from suitably qualified investigators of any nationality for grants, scholarships, or fellowships for research on leukaemia and allied conditions in the academic year 1955–6. Candidates with programmes of research on any aspect of malignant disease which may throw incidental light on problems of leukaemia will be eligible for consideration. Full details and forms of application (to be returned before March 31) may be obtained from the secretary of the (European) Scientific Advisory Committee, Lady Tata Memorial Trust, 38, Old Queen Street, London, S.W.1.

Grants are available for research expenses or to provide scientific or technical assistants for senior workers. Scholarships (£600 per annum for whole-time research, with proportional adjustment for part-time work) are awarded for the personal remuneration of workers carrying out approved research under suitable direction. In addition one or more Lady Tata Memorial Research Fellowships (£1,000 per annum) may be awarded to workers with considerable research experience.

N.A.P.T. Scholarships.—The National Association for the Prevention of Tuberculosis offers a Hunter scholarship of £350 to enable a graduate of a British medical school who qualified between the years 1936 and 1951 inclusive to study tuberculosis abroad. The association also offers two Hunter scholarships of £150 each for medical students who have developed tuberculosis during their studies. Full details from N.A.P.T., Tavistock House North, Tavistock Square, London, W.C.1. The last date for applications is February 28.

Royal College of Surgeons.—At the council meeting on January 13 Dr. G. J. CUNNINGHAM, senior lecturer in morbid anatomy and histology at St. Bartholomew's Hospital, was appointed Sir William Collins Professor of Human and Comparative Pathology in the College. Dr. JOHN VANE was appointed senior lecturer in the department of pharmacology.

College of General Practitioners.—The Scottish Council of the College of General Practitioners was constituted at a meeting held in Edinburgh on January 13. The members of the Council are: Drs. D. PEEBLES BROWN (Kilmalcolm), MARY ESSLEMONT (Aberdeen), C. M. FLEMING (Edinburgh), W. W. FULTON (Glasgow), W. S. GARDNER (Glasgow), I. D. GRANT (Glasgow), J. M. HENDERSON (Pitlochry), G. W. IRELAND (Pathhead), LOWELL LAMONT (Edinburgh), K. A. MACKAY (Laggan), T. S. MCDONALD (Glasgow), J. G. MUNRO (Beauly), DAVID MYLES (Forfar), R. A. B. RORIE (Dundee), C. S. SANDEMAN (Durness), RICHARD SCOTT (Edinburgh), J. A. SHEARER (Bucksburn), J. M. STALKER (Dundee), GEORGE SWAPP (Aberdeen). The office bearers appointed were: Dr. J. M. HENDERSON (*Chairman*), Dr. LOWELL LAMONT (*Vice-chairman*), Dr. RICHARD SCOTT (*Honorary Secretary and Treasurer*). The Scottish Office of the College is situated at Livingstone House, 39, Cowgate, Edinburgh, 1.

Refresher Courses for School Medical Officers.—In a memorandum to local education authorities (No. 489, dated January 19), the Minister of Education draws attention to the Postgraduate Medical Federation's refresher courses for general practitioners. "The Minister," says the memorandum, "is prepared to consider for grant expenditure incurred by local education authorities on enabling school medical officers to attend the refresher courses, provided that it is allowed by the district auditor at audit." Details of the courses may be obtained from the Postgraduate Medical Federation, 3, Gordon Square, London, W.C.1.

British Association of Plastic Surgeons.—At the annual general meeting in December the following officers and council were elected for 1955: *President*, Professor T. POMFRET KILNER (Oxford) (*previously president in 1948*); *hon. treasurer*, Mr. R. P. OSBORNE (Liverpool); *hon. secretary*, Mr. J. P. REIDY (London); *council*, Mr. J. N. BARRON (Salisbury), Mr. W. HYNES (Sheffield), Mr. P. H. JAYES (London), Mr. D. N. MATTHEWS (London) (*immediate past-president*), Mr. RAINSFORD MOWLEM (London), Mr. MORTIMER SHAW (Leeds), Mr. J. S. TOUGH (Glasgow), and Mr. A. B. WALLACE (*editor*) (Edinburgh).

Research on Cerebral Palsy.—The Nuffield Foundation has made grants totalling £17,500 to support a 5-year programme of research at Frenchay Hospital, Bristol, into the neuropathology of cerebral palsy and mental defect. The work is sponsored by the South-Western Regional Hospital Board and the Burden Mental Research Committee, as part of their programme of investigations on mental deficiency, and will be directed by Dr. R. M. NORMAN, lecturer in neuropathology at Bristol University, assisted by Dr. H. URICH. It is intended to examine the brains of those dying from cerebral palsy whose early history and clinical features have been adequately recorded, and a new laboratory is being equipped for this purpose. It is hoped that, when the new laboratory is in operation, material will be sent for detailed neuropathological examination not only from local hospitals but also from hospitals in other regions.

Hull Phonodiary.—Last year (October 2, p. 807) we gave the text of a gramophone record on cancer which was made available to the public over the Hull telephone system. A second record has now been prepared dealing mainly with the method of treatment and the aftercare of cancer patients. The first record resulted in over 5,000 telephone inquiries, at the rate of about 400 a week. This experiment in cancer education is sponsored by the Marie Curie Memorial Foundation.

Family Doctor.—The February issue tells readers how to avoid 'flu and what to do if they do get it. A new departure is headed "*Family Doctor* brings you a chance to listen in." This is selected extracts from a recent article in the *Practitioner*. The idea behind the title is that in the medical journals doctors write for each other, but sometimes they say things "that parents would like to overhear." The subject of the article is "Health and Normality in Childhood." Other articles include the career profile of a remedial gymnast, "How to tell if it *is* heart disease" by Professor Walter C. Alvarez, and advice on dysmenorrhoea.

COMING EVENTS

Milroy Lectures.—Dr. J. A. SMILEY will deliver the Milroy Lectures at the Royal College of Physicians, Pall Mall East, on February 8 and 10, at 5 p.m. His subject is "Personal Factors in Accident Proneness."

Fédération Internationale Pharmaceutique.—The sixteenth assembly will be held at the Senate House of London University from September 19 to 23. There will be symposia on "The Pharmacy of Blood, Blood products, and Blood Substitutes," "Recent Advances in Pharmaceutical Analysis," "Ion Exchange," and "Tests for the Sterility of Pharmaceutical Preparations." Fuller details from the secretary of the organizing committee, 17, Bloomsbury Square, London, W.C.1.

NEW ISSUES

British Journal of Ophthalmology.—The new issue (Vol. 39 No. 1) is now available. The contents include:

OCULAR CHANGES IN ACUTE SYSTEMIC LUPUS ERYTHEMATOSUS. F. Clifton and C. H. Greer.
SIGNIFICANCE OF CONGENITAL CYCLO-VERTICAL MOTOR DEFECTS OF THE EYES. A. Urrets-Zavalia.
CORNEAL ULCERATION. SENSITIVITY OF BACTERIA TO ANTIBIOTICS. Tulsi Das, M. L. Sur, and M. R. Chaddah.
DEGENERATIONS OF THE DOG RETINA. VII. CENTRAL NON-PROGRESSIVE DEGENERATION DUE TO AN ANOMALY OF THE GANGLION CELLS AND THEIR AXONS. H. B. Parry.
EFFECTS OF COCARBOXYLASE ON THE DEVELOPMENT OF GALACTOSE CATARACT IN RATS. R. Van Heyningen, A. Pirie, and J. Blackwell.
MULTIPLE MYELOMATOSIS WITH PROPTOSIS. A. Handousa.
CONGENITAL ECTODERMAL DYSPLASIA. I. D. R. Gregory.
UNUSUAL CASE OF MYASTHENIA GRAVIS. I. Lloyd.
INTRA-OCULAR MAGNETIC AIR-GUN SHOT. V. P. S. Tomar.
SIMPLIFIED NEAR POINT RULE. J. Foster.
MEASURING STRABISMUS HOOKS. A. B. Moffatt.

Issued monthly; annual subscription £4 4s.; single copy 8s. 6d.; obtainable from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

British Journal of Industrial Medicine.—The new issue (Vol. 12, No. 1) will be available in a week's time. The contents include:

RESEARCH IN INDUSTRIAL HEALTH IN THE CHEMICAL INDUSTRY. M. W. Goldblatt.
MANGANESE POISONING IN MOROCCAN MINERS. J. Rodier.
DERMATOSES IN JUTE WORKERS. John Kinnear, John Rogers, and Alexander Mair.
TALCOSIS OF UNUSUALLY RAPID DEVELOPMENT. G. P. Alivisatos, A. E. Pontikakis, and B. Terzis.
RESPIRATORY TRACT INJURY CAUSED BY CERTAIN ISOCYANATES USED IN PRODUCTION OF LACQUERS. Ake Swensson, Carl-Eric Holmqvist, and Karl-David Lundgren.
DIMETHYL SULPHATE POISONING. T. R. Littler and R. B. McConnel.
VANADIUM POISONING FROM GAS TURBINES. R. C. Browne.
THE TOXICITY OF OZONE IN THE PRESENCE OF OXIDES OF NITROGEN. W. M. Diggle and J. C. Gage.
THE CONSTRUCTION OF CRITICAL ORIFICES WORKING WITH PRESSURE DROPS OF LESS THAN HALF ATMOSPHERIC PRESSURE AND THEIR USE IN CONTROLLING AIRFLOW. H. A. Druett.
THE HEALTH HAZARDS OF THE SENIOR EXECUTIVE. A. R. Cooper.
THE HEALTH OF THE INDUSTRIAL WORKER IN IRAQ. A. Michael Critchley.

Issued quarterly; annual subscription £2 2s.; single copy 12s. 6d.; obtainable from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Tuesday, February 1

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Dr. E. F. Gale: Actions of Antibiotics on Bacteria.
INSTITUTE OF DERMATOLOGY.—Fifth of a series of semi-permanent exhibitions from February 1 to 25, by Dr. R. T. Brain, on Vascular Naevi (Haemangiomata).
ROYAL MEDICAL SOCIETY, Edinburgh.—8 p.m., clinical meeting.
ST. MARY'S HOSPITAL MEDICAL SCHOOL.—At Wright-Fleming Theatre, 5 p.m., Mr. A. W. Bourne: "Functional" Uterine Haemorrhage.

Wednesday, February 2

EDINBURGH CLINICAL CLUB.—4 p.m., visit to Department of Radiotherapy, Edinburgh Royal Infirmary.
INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. J. O. Oliver: Bacteriology of Skin Diseases.
INSTITUTE OF LARYNGOLOGY AND OTOLOGY.—6 p.m., Professor M. Arslan (Padua): Effects of Ultrasonic Waves on the Ear.
MIDLAND MEDICAL SOCIETY.—At Birmingham Medical Institute, 154, Great Charles Street, 8.15 p.m., Mr. A. Ruscoe Clarke: Blood Loss and Blood Transfusion in Civilian Trauma.
OXFORD UNIVERSITY MEDICAL SOCIETY.—At Radcliffe Infirmary, 8.15 p.m., Professor H. A. Krebs, F.R.S.: Role of Biochemical Concepts in Medicine.
ST. MARYLEBONE HOSPITAL FOR PSYCHIATRY AND CHILD GUIDANCE.—5.30 p.m., Dr. Doris M. Odlum: Relationship of Psychiatry to General Medicine.
YORKSHIRE SOCIETY OF ANAESTHETISTS.—At General Infirmary at Leeds, 8.30 p.m., Dr. G. S. W. Organe: Abuse of Tracheal Intubation.

Thursday, February 3

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Professor P. B. Medawar: Tissue Transplantation Immunity.
FACULTY OF HOMOEOPATHY.—At Royal London Homoeopathic Hospital, 5 p.m., Dr. W. Lees Templeton and Dr. C. O. Kennedy: Provings of A.C.T.H. and Cortisone, Including Investigation of Bowel Flora; Dr. L. R. Twentyman: Observations on Iscador Treatment of Cancer.
INSTITUTE OF PSYCHIATRY.—5 p.m., Professor H. J. Eysenck: Psychology and the Foundations of Psychiatry.
LIVERPOOL MEDICAL INSTITUTION.—8 p.m., Dr. V. K. Summers: Syndrome of Hyperthyroidism; with a note on radioactive iodine tracer studies by Dr. W. Kulke.
NUFFIELD ORTHOPAEDIC CENTRE.—At Wingfield-Morris Orthopaedic Hospital, 8.30 p.m., Dr. E. G. L. Bywaters: Natural History of Rheumatoid Arthritis.
ROYAL ARMY MEDICAL COLLEGE.—5 p.m., Professor C. G. Rob: Preservation and Transplantation of Human Blood Vessels (with lantern slides).
ROYAL COLLEGE OF SURGEONS OF ENGLAND.—5.30 p.m., Otolaryngology Lecture by Sir Gordon Gordon-Taylor: Swellings of the Neck and their Management.
ST. ANDREWS UNIVERSITY.—At Physiology Department, Medical School, Small's Wynd, Dundee, 5 p.m., Dr. T. Anderson: Unwanted Effects of Chemotherapy.
ST. GEORGE'S HOSPITAL MEDICAL SCHOOL.—5 p.m., Sir Paul Mallinson: psychiatry demonstration.

Friday, February 4

●INSTITUTE OF DERMATOLOGY.—5.30 p.m., clinical demonstration by Dr. P. J. Hare.
INSTITUTE OF DISEASES OF THE CHEST.—5 p.m., clinical demonstration by Mr. V. C. Thompson: Tumours of the Lung—II.
●LONDON SCHOOL OF HYGIENE AND TROPICAL MEDICINE.—6.30 p.m., Dr. J. A. Scott: Health.
POSTGRADUATE MEDICAL SCHOOL OF LONDON.—4 p.m., Professor R. E. Tunbridge: Clinical and Histochemical Study of some of the Disorders of Connective Tissue.
●ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW.—5 p.m., Mr. J. Sloan M. Robertson: Paralysis as a Surgical Problem.
ROYAL MEDICAL SOCIETY, Edinburgh.—8 p.m., Sir James Learmonth: Some Aspects of Surgery Elsewhere.
WHIPPS CROSS HOSPITAL MEDICAL SOCIETY.—8.30 p.m., Mr. B. Fairburn: Surgical Treatment of Cerebral Vascular Catastrophes.

Saturday, February 5

●INSTITUTE OF ORTHOPAEDICS.—February 5 and 6, week-end course for Registrars.
MIDLAND TUBERCULOSIS SOCIETY.—At Birmingham Chest Clinic, 3 p.m., Mr. D. M. E. Thomas: Surgical Treatment of Primary Tuberculosis.

APPOINTMENTS

LONG, M. C. W., M.B., B.S., Deputy Treasury Medical Adviser.
MUNRO, T. A. H., M.D., F.R.C.P.Ed., D.Psych., Physician Superintendent, Royal Edinburgh Hospital for Mental and Nervous Disorders, as from November, 1954.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Lynn.—On January 15, 1955, to Constance, wife of Dr. C. R. Lynn, of 25, Elgin Avenue, Harold Park, Essex, a son—Jonathan.
Weber.—On December 29, 1954, at St. Bartholomew's Hospital, London, E.C., to Rosalie, wife of Dr. G. N. Weber, a son—Jonathan Norden.

DEATHS

Morton.—On January 3, 1955, in London, Harold James Storrs Morton, M.D., of Brinkley, Newmarket, Suffolk, formerly of Bridlington, Yorks, and St. John's Wood, London, N.W., aged 68.
Rickard.—On October 4, 1954, at "The Firs," Simons Town, South Africa, Cecil Rodney Rickard, M.R.C.S., L.R.C.P., Surgeon Captain, R.N., retired.
Smith.—On January 4, 1955, at St. Andrews, John Alfred Macdonald Smith, M.B., F.R.C.S.Ed., late of Kalimpong, Bengal, India.
Steavenson.—On January 3, 1955, Charles Stanley Steavenson, M.B., Ch.B., of Felix House, Middleton St. George, Co. Durham.
Taylor.—On January 6, 1955, at Downs Cottage, 76, Northwood Road, Tankerton, Kent, Frederic Cecil March Taylor, M.R.C.S., L.R.C.P., D.P.M., aged 55.

Any Questions ?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Vaccination in Pregnancy

Q.—*Is pregnancy a contraindication to the revaccination of a woman (successfully vaccinated as an infant) who now wishes to go abroad? Is primary vaccination contraindicated?*

A.—Vaccination is often stated to be harmless to both the pregnant woman and her foetus. However, MacArthur,[1] who analysed the results of a questionary from the West of Scotland, concluded that vaccination during the first trimester of pregnancy increased the risk of abortion; damage to the foetus did not occur after vaccination in the later months. Notwithstanding these findings, severe vaccinia, like any other acute infection, might endanger the foetus at any stage of development. It would seem wise, therefore, not to vaccinate during pregnancy unless there is a likelihood of exposure to smallpox, or good reason to expect the development of a vaccinoid or an immediate reaction rather than a typical primary "take." The decision in the present case depends on circumstances; the risks of revaccination after the third month of pregnancy are certainly not great, and postponement of vaccination till after the patient's confinement may not be feasible. Primary vaccination in pregnancy is more likely than revaccination to endanger the life of the foetus. Nevertheless, in the face of smallpox, all vaccination risks must be accepted.

REFERENCE

[1] *Lancet*, 1952, **2**, 1104.

Treatment of Hypogonadism

Q.—*A man of 30 with slight hypogonadism has been treated for three months with methyl testosterone, 100 mg. a day, with some success. Is there any danger in this treatment? Is further improvement likely if treatment is continued? To what extent can the improvement be expected to be maintained without further treatment?*

A.—In my experience eunuchs can be maintained on a dose of only 25 mg. of methyl testosterone daily, so that a dose of 100 mg. daily for a person with slight hypogonadism seems somewhat high. A daily dose of 100 mg. is unlikely to inhibit pituitary function, but it may cause slight oedema as a result of salt and water retention; this disappears on cessation of therapy, so there is no real danger in this treatment. Improvement in hypogonadism treated with testosterone occurs over a period of six to nine months or more depending on the gravity of the condition. As therapy is merely a replacement of a deficiency, relapse can be expected if treatment is stopped.

Technique of Artificial Insemination

Q.—*Could you please give me, briefly, the technique for artificial insemination?*

A.—Artificial, or what is better termed therapeutic, insemination can be carried out in various ways. All of them are done at what is judged to be the ovular phase of the menstrual cycle. Intracervical insemination is the commonest technique. To perform this, the cervix is exposed with a speculum and the seminal fluid injected into the cervical canal by means of a suitable syringe and cannula. For intrauterine insemination, the cannula is passed through the internal os and about 0.25 ml. of semen injected into the uterine cavity. After the injection the woman should remain with her legs together and her pelvis slightly elevated for 10 to 30 minutes. A further alternative is to place a cervical cap, into which the seminal fluid has been introduced, over the cervix, where it remains for 12 hours, the woman having been instructed to remove it at the end of this time.

Where, by reason of impotence or through some mechanical cause, it is impossible for the husband to ejaculate deeply within the vagina, the technique of "self-insemination" may be recommended. For this, the husband produces semen by masturbation into a clean dry glass jar at the ovular phase of the cycle. The seminal specimen is taken up into a special self-insemination syringe (designed by Dr. Joan Malleson and supplied by Messrs. Allen and Hanburys) which the wife then inserts into the vagina to its full extent. By pressing home the plunger the semen is deposited in the place where effective coitus would have left it. If necessary, a non-greasy and non-spermicidal lubricant should be applied to the outside of the syringe before it is inserted. After use, the syringe should be washed in warm soapy water and dried. Sterilization is unnecessary.

Transfusion Problems in a Small Hospital

Q.—*Working in a small maternity hospital, I am required occasionally to give blood transfusions. Only group O blood (Rh positive and negative) is stocked here. Group B and group AB blood are almost impossible to obtain. Group A blood is kept at a neighbouring hospital; if this is used it often results in wastage of group O stock. The cross-matching technique is extremely primitive: patient's serum, donor cells from the bottle, and normal saline are mixed and left for 15 minutes on a slide. There is no incubator available. Will you therefore please answer the following questions? Is the risk of incompatible transfusion materially increased by the giving of group O blood to patients of other groups? Is it justifiable to use group O blood to avoid wastage in a small blood bank? Does previous childbirth or transfusion affect the issue? (It is realized that the correct Rhesus type must be used.) Is a cold saline cross-match considered adequate for non-urgent transfusion?*

A.—The administration of group O blood to patients of other groups does not, as a rule, give rise to haemolytic reactions. In rare cases—for example, when half or more of the recipient's blood is replaced by group O blood—a reaction may occur; and if the donor has α or β haemolysins in his serum (as occurs after injection of concentrated antibody globulins) these lysins may destroy the recipient's cells.

The exclusive use of group O blood was probably arranged as the least dangerous procedure for a small unit with an "occasional transfusionist" in charge. It has been shown that haemolytic reactions usually result from blunders in ABO grouping by inexperienced or excited workers, or by confusion between two patients of similar name but different ABO group. If this source of error is eliminated, one is left with Rhesus antibodies and the host of irregular antibodies reacting with the antigens M, N, S, P, K, k, Fy^a and Fy^b and others, all of which antibodies are rare: apart from the common Rhesus antibody anti-D, the frequency of irregular antibodies is of the order of 1 in 3,000 recipients; the risk of such an antibody causing a haemolytic reaction is of this order, comparable with the incidence of haemolytic transfusion reactions without demonstrable cause.

Previous childbirth or transfusion makes such antibodies a little more frequent. The frequency of anti-D is, of course, greatly increased by pregnancy or Rh-incompatible transfusion, but the other antigens are weak and rarely initiate the production of antibodies: if one is concerned only with urgent transfusions it is reasonable to subject the patient to this approximately 1 in 3,000 risk of a haemolytic reaction. Even if a reaction does occur, the risk of death is only about 1 in 15, so the overall risk is about 1 in 45,000—a risk far less than that incurred by leaving an exsanguinated patient for two or three hours. Even so, many transfusions can be avoided by the free use of dextran.

There is some argument about the standard of cross-matching which should be adopted for non-urgent transfusions. The expert can use almost any method he likes with safety, but the occasional transfusionist should adopt one of the standard tube methods, since he is less likely to make mistakes with them than with any other. I would suggest a cross-match in saline at about 20° C. (room temperature) and by the Sheffield technique in albumin, at 37° C. A 37° C. incubator and a small centrifuge are essential, and no adequate blood grouping can be done without them. The recommended procedure is:

Place two tubes in each of two racks, labelling one of each pair "test," the other "control." Into each "test" place 3 drops of recipient's serum, into each "control" 3 drops of saline.

Into each tube place 3 drops of an approximately 5% suspension in saline of donor cells which have been removed from the bottle or pilot bottle with a sterile Pasteur pipette and washed once in saline. Leave one rack at room temperature; place the other in the incubator for 1½ hours. After 1½ hours remove from the incubator without shaking, and remove as much as possible of the supernatant serum and saline: replace it with 6 drops of 20% bovine albumin solution without disturbing the sedimented cells. Return to the incubator for half an hour. At the end of a further half-hour—that is, two hours from the beginning—remove samples from the tubes and examine microscopically.

If the tubes marked "test" show even a few clumps of cells, while those marked "control" show more, then the blood should be regarded as incompatible. A number of samples known to be incompatible should be tried at leisure, using smaller proportions of serum, so that the appearance of incompatibility becomes familiar.

But would it not be possible to send all non-urgent transfusions to a large local hospital or to the regional depot for cross-matching? Were I the writer of this question, I should be extremely worried by the lack of facilities, and one of my first actions would be to ask the regional transfusion director to call and advise me on the best use of the facilities which were available. It is definitely not justifiable for an "occasional transfusionist" to use a saline tile cross-match method for "non-urgent" transfusions.

Atropine and Neostigmine after Relaxants

Q.—*It is customary when counteracting relaxants at the end of an operation to give intravenous atropine some minutes before intravenous neostigmine. Is there any experimental evidence to suggest that the speed of action of these two drugs is in fact different, so that they may not be given together?*

A.—The practice of administering atropine some 10 minutes before neostigmine, when reversing the effects of a curare-like drug, is an attempt to avoid the coincidence of any initial bradycardia due to atropine and the bradycardia due to neostigmine. It is certain that a brief bradycardia generally precedes tachycardia when atropine is administered hypodermically. But there is some doubt whether this happens when the drug is injected intravenously. Indeed, one author[1] is emphatic that it does not. There is therefore some experimental evidence to suggest that the danger of giving atropine and neostigmine together is not a great one.

Any difference in the speed of action between atropine and neostigmine is probably not an important consideration in this connexion, since both take about five minutes to reach their maximum effect when given intravenously.[1,2]

REFERENCES
1 Hunter, A. R. (1953). *British Medical Journal*, 1, 640.
2 Shearer, W. M. (1951). *Anaesthesia*, 1951, 6, 76.

NOTES AND COMMENTS

Fetishism.—Dr. DORIS ODLUM (London, W.9) writes: I have read with interest the answer to the question on fetishism ("Any Questions?" January 15, p. 178). In the course of the answer, however, it is stated that this appears not to occur in women, but from my own clinical knowledge I can state that in fact it does do so. One of my patients—a woman in the forties of the highest character and a teacher of English—was deeply disturbed by feelings of guilt because she had a definite orgasm whenever she saw a pair of eye-glasses on a table or in any place where light was reflected on them. She found it a great temptation to put glasses in such a position and from time to time did in fact do so, but her most satisfactory orgasm came when the situation arose by chance. In another case a woman had strong sexual sensations whenever she touched certain types of fur, notably sealskin, moleskin, or squirrel, or when she handled certain types of velvet. In another case orgasm always resulted from the sight of a riding whip or cane, associated with masochistic fantasies. None of these cases was suffering from any kind of emotional disturbance and they were all making a good adjustment to life. I have also known of other cases where the women were happily married and were all well adjusted both to their husbands and children and to life in general. The condition is not necessarily due to any morbid tendency produced by lack of emotional satisfaction, but rather represents an alternative or supplementary form of sexual gratification. I have also had cases where some girl children as well as boys had obvious orgasms when they were sucking a blanket before going to sleep and who insisted on being allowed to suck the blanket before they would settle down at night, and a number of children of both sexes show obvious evidence of rudimentary orgasm when they smack or hug a doll or teddy bear. I have not heard of a case in which a woman has gone so far as to practise fetishism in public, and in the cases that I know of they are extremely loath to speak of it either because they feel guilty about it or because they realize that it would give rise to moral and social condemnation even though they themselves did not feel any guilt. This may be the reason why its existence has not been generally recognized.

Drugs and Fertility.—Mr. HOWARD G. HANLEY (London, W.1) writes: The answer to the question on this subject ("Any Questions?" January 8, p. 118) states, ". . . there seems good evidence that the sulphonamides in therapeutic doses may suppress temporarily the development of spermatozoa within the testis and so lead to impaired fertility." I have been unable to find the original reference in support of this belief, but have accumulated some evidence to show that it is untrue. I have carefully followed up 21 cases of pyospermia treated with sulphathiazole, "sulphatriad," or "gantrisin" in large and sometimes prolonged dosages, and, although one can expect the sperm motility to improve when the pus cells are cleared, in no case has the count been reduced or impaired in any way. The two cases which have been referred to me as showing reduced spermatogenesis following sulphonamide therapy both gave a clear history of a relatively severe fever (influenza with quinsy; lymphangitis) which necessitated the giving of the sulphonamides, and I suspect that it was the hyperpyrexia or the debility, not the sulphonamides, which were responsible for the temporarily impaired fertility. I also suspect that smoking has been blamed in the same unscientific manner, and whether spermatogenesis can be depressed by nicotine *per se* rather than by the various pathological disturbances attributed to excessive smoking is a debatable point. The opening sentence of the answer to the question contains the crux of the matter, "There have been few, *if any* (my italics) systematic studies of the effect of drugs on fertility, and consequently knowledge on this subject is scanty."

OUR EXPERT writes: Mr. Hanley's comments may well be quite justified. The first sentence of my answer to which he draws attention was indeed intended to contain the crux of the matter.

Corns Between the Toes.—Dr. C. GUY MILLMAN (Morpeth, Northumberland) writes: May a psychiatrist get down to earth and suggest that animal wool is preferable to the gauze advised in the reply to the question about corns between the toes ("Any Questions?" January 1, p. 56)?

Correction.—In the *Journal* of January 8, p. 111, it was recorded in error that the University of Oxford had granted leave of absence to Professor R. R. Macintosh for three months from January 1, the decree having been withdrawn before Congregation on November 30, 1954.

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